South African Medical Journal
Suid-Afrikaanse Tydskrif vir Geneeskunde

Vol. 24, No. 38 Cape Town, 23 September 1950 Weekly 2s

PRIMARY AMYLOIDOSIS

REPORT OF A CASE AND A REVIEW OF THE LITERATURE

SYLVIA GOLDBERG, M.B., CH.B., B.SC. HONS. (RAND.)
Department of Pediatrics, Coronation Hospital, Johannesburg

Amyloidosis as a complication of chronic diseases such as tuberculosis and syphilis or of prolonged suppurative processes is a well-recognized condition. Only in recent years, however, has attention been drawn to a lesser-known type, the so-called primary form where there is no apparent etiologic factor to account for the amyloid deposition.

The first account of primary amyloidosis is variably ascribed to Wilks (1856) or Wild (1886). Following a report of three such cases in 1929 by Lubarsch, several reports of primary systemic amyloidosis have appeared, mainly in the American literature. Some of these have accompanied comprehensive reviews of the literature on the subject. Dahlin (1949), in a recent, very full analysis gives the number of cases recorded up to the time of his paper as 57. He himself presents six cases. Autopsy reports were available in 60 of the cases reviewed by him.

CASE REPORT

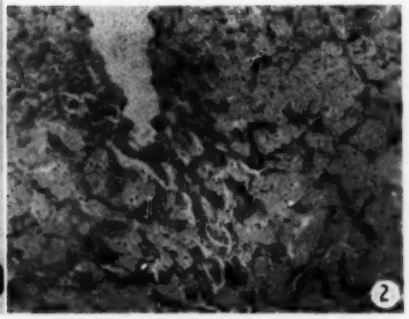
A male Msutu child aged nine years was admitted to the Coronation Hospital on 23 May 1949. The history was obtained from the grandmother. There had been progressive swelling of the abdomen for two months. Minor complaints were a slight, unproductive cough and painful feet, both of one week's duration. At no time had there been any swelling of hands, feet or face. His urinary output was normal, although he had always had nocturia.

Despite a good appetite, he was losing weight. There were no significant details in the past history. The child was born on a farm near Bechuanaland where he had lived all his life and had had an apparently adequate mixed diet. The child's physical and mental development seem to have been normal.

Examination revealed an intelligent, co-operative child, somewhat thin and under-sized for his age. The presenting feature was abdominal enlargement. Palpation revealed marked hepatomegaly, maximal in the mid-line where the liver edge was felt at the level of the umbilicus (Fig. 1). The surface was firm, uniform and non-tender, and the edge firm and thin. There was no free fluid in the abdomen. Other findings were two small palpable glands in the left mid-cervical chain, clinical cardiac enlargement to the left and a long systolic murmur heard all over the praecordium. There was an impaired percussion note with diminished air entry at the right base.

Fig. 1. The patient. The outline of the liver has been marked out with chalk.

Fig. 2. Microscopic section from a liver biopsy showing the extensive amyloidosis.



Examination of the central nervous system revealed no abnormality.

Investigations. Vollmer patch test: Negative on admission and dubious when repeated two month's later.

Haemoglobin, 8.5 gm. %.
Colour Index, 0.76.
Erythrocytes, 3.6 million per c.mm.
Leucocytes, 14,000 per c.mm.
Neutrophils, 68%; Monocytes, 5%; Lymphocytes, 27%.
Erythrocyte Sedimentation Rate, 47 mm. per hour. Packed cell volume 30%, the corrected sedimentation rate falling in the zone of a slight increase.
No malaria parasites were detected in the blood smears.

The modified Ide test for syphilis was negative.
Blood urea, 37 mg. per 100 c.c.
Serum albumin, 3.5%; serum globulin, 3.8%.
Blood cholesterol, 100 mg. per 100 c.c.
Thymol turbidity test, 1.5 units.
Thymol flocculation test, Negative.
Takata ara reaction (Ucko's modification), 2+.
Icteric Index, 4; van den Bergh reaction, Negative.
Alkaline phosphatase, 10.4 units (King-Armstrong).
Prothrombin index, 108%.

The urine contained no albumin, bilirubin, urobilin or urobilinogen and microscopic examination showed occasional granular and hyaline casts and triple phosphate crystals. No reducing substances, acetone or diacetic acid were found in specimens of urine taken at intervals during 24 hours. Amino acids were not detected on chromatographic analysis of the urine nor did the urine contain Bence-Jones protein. Bilharzia ova were not detected in the sediment of 24-hour specimens of urine.

Animal parasites including amoebae were not detected in the stools and no pathogenic bacteria were isolated. Numerous examinations of stool and gastric juice for *M. tuberculosis* were negative.

Glucose tolerance tests repeated on several occasions gave varying results. The fasting blood sugar was always 90 mg. per 100 c.c. On the first occasion this rose to 300 mg. per 100 c.c., after ingestion of 35 gm. of glucose, returned to resting level at two hours and rose again to 105 mg. per 100 c.c. at 2½ hours. On the second occasion there was no rise at all. On the third occasion there was a rise to 130 mg. per 100 c.c., a return to resting level at two hours and a secondary rise to 100 mg. per 100 c.c. at 2½ hours.

X-ray examination of the chest on admission showed some compression of the lower lobes of the lungs due to the large liver. There was no cardiac enlargement.

No abnormal changes were detected in the skull or long bones.

Barium meal examination revealed no organic lesion in the oesophagus, stomach or duodenum but the 1½-hour film showed clumping of the barium in the small bowel. No pathology was noted in the large bowel. An intravenous pyelogram, although technically not satisfactory, revealed no pathology in the renal tract.

The electrocardiogram was normal except for a high $QT/\sqrt{RR}$ ratio of 0.45.

Sigmoidoscopy of the lower 23 cm. of the large bowel did not reveal any ulceration or other obvious pathology.

During his hospital stay, the child had several blood-streaked stools and repeated epistaxes. Shortly after admission he suffered what appeared to be an episode of measles with pyrexia, rash, conjunctivitis and generalized lymphadenopathy, complicated by a left basal pneumonia. Radiographic examination of the chest had revealed no pathology on admission but now showed an area of consolidation in the left lower lobe which appeared compressed with partial atelectasis. As the child improved clinically, radiological improvement and complete resolution took place.

Two diagnoses were entertained. The first was that of glycogen storage disease, and the second of hepatic cirrhosis giving rise to portal back pressure and intestinal haemorrhage. Owing to the non-contributory laboratory data, a liver biopsy was performed under local anaesthesia. Sections of the specimen obtained were stained with haematoxylin and eosin, methyl violet, and Best's carmine. On examination they revealed a fairly extensive amyloidosis affecting the parenchyma (Fig. 2). An intravenous Congo red test was carried out on the basis of 0.25 c.c. of dye per kg. body weight as described by Bray (1946). The result showed that there was moderate concentration of the dye in the 4-minute specimen of serum but none was detectable in the one-hour specimen and none was detected in the urine.

After a stormy passage with haemorrhage following the liver biopsy, the child did very well apart from intermittent epistaxes and blood in the stools. He gained weight and remained apyrexial for a period of observation extending over six months. Serial X-ray examination of the chest revealed no pulmonary pathology.

He is still under observation but is seen at unavoidably irregular intervals.

In view of the reports of amyloidosis in association with Hodgkin's disease (Bannick *et al.*, 1933), a cervical gland biopsy was done. The following is the pathologist's report:—

'Section of this cervical lymph gland shows marked sinus catarrh and chronic inflammatory stimulation. In addition, foci of chronic granulomatous change in which there is reticulo-endothelial and multinucleated giant cell reaction are present. The granulomatous lesions have a tendency to fibrose; no central caseation has been observed and acid fast bacilli have not been found in suitably stained preparations but the histological features are consistent with tuberculous lymphadenitis' (Dr. J. F. Murray).

DISCUSSION

In the earlier reports and reviews of primary amyloidosis considerable importance was attached to the difference in the distribution and staining properties of the amyloid in the primary and secondary types of the disease. The classification of amyloidosis used by Koletsky and Stecher (1939) was generally employed, viz:—

(a) Secondary amyloidosis, associated with prolonged suppuration, tuberculosis and syphilis.
(b) Primary amyloidosis.
(c) Amyloidosis associated with myelomatosis.
(d) Tumour-forming amyloidosis.

Stress was laid on the features characterizing the secondary type, namely the involvement of liver, spleen, kidneys and adrenals, and the typical staining reactions of the amyloid with Congo red and methyl violet. As distinct from this form, the primary type was characterized by the absence of known etiologic factors, and the distribution of the material in mesodermal tissue, smooth and skeletal muscle, the cardio-vascular system and the gastro-intestinal tract, leaving uninvolved the organs usually affected in secondary amyloidosis. In addition, the amyloid might give variable and atypical staining reactions. The amyloidosis associated with myelomatosis has a distribution similar to the primary form. The tumour-forming type is usually of the primary form or associated with multiple myelomata and is characterized by the formation of small or large, solitary or multiple tumours.

With increasing numbers of reports however, it is being realized that there is in point of fact considerable overlap in the features previously thought to differentiate the two forms of the disease. For example, Dahlin (1949) in his review of 57 cases, 45 of which were autopsied, considered cardiac amyloid infiltration to be one of the most constant features of the primary form of the disease. It is specifically mentioned in 54 instances and regarded as the cause of death in 25 cases. Lindsay (1946) however, in a comprehensive survey of cardiac amyloidosis, quotes reports by various authors which demonstrate that it occurs not uncommonly in secondary amyloidosis. Thus, myocardial involvement was found in cases of amyloidosis secondary to syphilis, renal infection and bronchiectasis.

Conversely, in the primary cases described, the amyloid may be deposited in organs commonly involved in the secondary forms. Dillon and Evans (1942), Zemp (1947), Brown and Selzer (1944) and Bannick *et al.* (1933) have all reported cases of primary amyloidosis in which the amyloid was deposited in liver, spleen, kidneys and adrenals.

Dahlin (1949) considers that of all the criteria for the diagnosis of primary systemic amyloidosis, absence of pre-existing etiologic disease is the only constant clinical and pathological finding, and that neither the staining reactions nor the distribution of the amyloid is so specific as to constitute a basis for distinction between the two types. He comments on the involvement of liver and kidneys in 21 cases and of the spleen in 20 cases of primary amyloidosis.

In the case presented here, extensive radiological and laboratory investigations failed to reveal any etiological factor for the amyloid deposition with the exception of a possible small tuberculous gland. Although it is most unlikely that the minimal changes in the gland could be the causative factor in an extensive hepatic amyloidosis, it is possible that the gland is but an indication of a more wide-spread tuberculous process. However, this possibility was considered a very remote one after exhaustive studies had failed to reveal any other focus. In addition, it must be remembered that the histological findings were only suggestive of tuberculosis, as neither caseation nor acid fast bacilli were observed.

We therefore present this as a case of primary amyloidosis. Such a diagnosis can only be made tentatively without the completeness of full autopsy findings and a follow-up study of the case is most important.

It is proposed to discuss this case by reviewing the symptoms and signs in relation to the literature on the subject, in addition to features of the disease described by previous authors and not manifested in this case.

This would appear to be the youngest case so far reported. It is the third case to be reported from this country, the first one being that of Brown and Selzer (1944) and the second being that of Woolf (1950). It is the first case to be described in a South African Bantu. The sex incidence of this relatively rare disease appears to be more or less equal; the average age incidence is 55 years, ranging from 15 to 80 years.

Hepatic involvement in primary amyloidosis is not uncommon, being described in 21 cases and such marked involvement as to clinically suggest primary liver disease has been recorded by several authors. One of the three cases presented by Bannick *et al.* (1933) gave a 2-year history of progressive indigestion, had ascites and a large firm liver, developed progressive jaundice in addition to persistent albuminuria and hypertension, and at autopsy there was marked hypertrophy of spleen and liver with generalized amyloidosis affecting the spleen, liver, supra-renals, myocardium, lymph nodes, kidneys and other organs. Of the six cases presented by Dahlin (1949) in two, liver symptoms and signs dominated the clinical picture. In one a clinical diagnosis of cirrhosis of the liver was made, and in the other, obstructive jaundice was the diagnosis. Similarly in this child the clinical picture was dominated by the marked hepatomegaly, although it will be noted that all laboratory tests for liver function, with the possible exception of the glucose tolerance tests, showed no apparent impairment of function.

Cardiac involvement is a most striking feature in the cases recorded. Of the 45 cases reviewed by Lindsay (1946), 23 showed clinical evidence of cardiac failure during the course of their illness and in 18 of the 43 fatal cases cardiac failure appeared to be the immediate cause of death.

Amyloidosis may cause cardiac failure in several ways (Lindsay 1946), depending on the site of deposition of the material in heart or lungs—involvement of the latter, giving rise to chronic cor pulmonale. Dahlin (1949), too, emphasizes the high incidence of cardiac involvement. This occurred in all six of his cases and cardiac failure was the cause of the main clinical manifestations of the disease and of death in four of his cases. Lindsay (1946) gives the electrocardiographic findings which were available in nine cases. In six, low voltage was a prominent feature; in one the P-R interval was slightly prolonged and arrhythmia was present in two cases; in three of Dahlin's (1949) cases, the QRS complexes were of low voltage.

This child presented no clinical evidence of cardiac insufficiency, but had a long apical systolic murmur which was unrelated to posture or exercise and which persisted after iron and repeated transfusions had restored his haematological picture to normal. As previously mentioned, the electrocardiogram was normal except for a high $QT/\sqrt{RR}$ ratio.

Involvement of the gastro-intestinal tract is mentioned in 33 of the 54 autopsied cases reviewed by Dahlin (1949). Five had serious haemorrhage from the gastro-intestinal tract. It was slightly to moderately involved in all six of his own additional cases. Golden (1945) presents an interesting case of a 66-year-old negress in whom laparotomy revealed extensive gastric amyloidosis with two pyloric ulcers. Autopsy revealed extensive deposition of Congo red which had been injected three hours before death, all along the intestinal tract. In two of the cases presented by Dillon and Evans (1942) the stools were positive to the guaic or benzidine tests and one case had frank blood in the stools terminally. In this case, no proof that the intestinal haemorrhage was due to amyloidosis was obtained, but the association with hepatic amyloidosis

and the clumping of the barium in the small bowel may be regarded as suggestive, in view of the normal findings on sigmoidoscopy and the repeatedly normal stool cultures.

The diffuse deposition of amyloid may lead to involvement of numerous other organs including the tongue, the skin, lymph nodes, thyroid, adrenal, pancreas, spleen, skeletal muscle, bones (Koletsky and Stecher, 1939) and the nervous system. In almost all the autopsy reports available, the extensive nature of the amyloid deposition is a most striking feature, although the extensive amyloidosis ultimately revealed is not clinically suggested, the picture being dominated by the involvement of the organ maximally affected. The case of primary amyloidosis presented by Bannick *et al.* (1933) showed supra-renal insufficiency clinically, and at autopsy there was extensive supra-renal amyloidosis. The adrenal cortex was involved in three of Dahlin's (1949) cases, the deposition being similar to that seen in secondary amyloidosis, but there were no clinical signs of adrenal insufficiency. In Brown and Selzer's case (1944) there was extensive involvement of the whole of the supra-renal cortex.

Renal involvement is common and may be associated with gross albuminuria, as in the cases presented by Zemp (1947), Bannick *et al.* (1933), Dillon and Evans (1942) and in two of Dahlin's (1949) cases. Renal amyloidosis is well reviewed by Brown and Selzer (1944) who draw attention to the fact that although the clinical picture has several of the features seen in lipoid nephrosis, it is the globulin fraction which is mainly lost in the urine.

Dostrovsky and Sagher (1941) and Michelson and Lynch (1934) emphasized the characteristic cutaneous lesions and Dahlin (1949) noted their presence in about a fourth of the cases he reviewed, but no skin lesions except purpura were observed clinically in his six cases. No skin lesions were observed in the case presented here.

Weber *et al.* (1937) reviewed the association of primary amyloidosis with macroglossia and placed considerable emphasis on this association, but Dahlin (1949) considers the diagnostic value of macroglossia to have been over-rated as it was observed in less than a third of the cases reported in the literature and was absent in five out of his six cases.

The diagnosis of amyloidosis must be made primarily by biopsy results and may be suggested by the intravenous Congo red test of Benhold. In secondary amyloidosis, according to Stemmerman and Auerbach (1944), 90-100% absorption of dye may be regarded as a positive result but false positives may occur where renal tubular damage is present. Minimal amyloidosis may be associated with a negative result. Lindsay (1946) records the results of the test in 10 cases of primary amyloidosis. In five cases the result was positive, the absorption at one hour being 60-100%; in five the results were considered negative, absorption at one hour being 35% or less. In the case reported here there was 100% absorption. The prognosis of primary amyloidosis is variable and its treatment empirical. Of the 45 cases reviewed by Lindsay (1946) only two were alive at the time the reports were made and the duration of life from the onset of symptoms varied from four months to 16 years.

Amyloidosis can be produced experimentally by long continued injection of vaccine, tuberculin or sodium caseinate. An interesting hypothesis of the pathogenesis of amyloidosis is suggested by Brown and Selzer (1944). They emphasize the fact that the experimental production of amyloidosis is associated with a raised serum globulin, and hyper-globulinaemia exists in those diseases which are considered to cause the secondary form of the disease. They quote Sabin that certain globulins are normally produced in the reticulo-endothelial system and suggest that primary amyloidosis may be due to a functional disturbance of the reticulo-endothelial system which is at first stimulated to increased globulin production but is later unable to handle these products, which are then deposited in the surrounding tissues as amyloid. They are struck by the association of amyloidosis with diseases of the reticulo-endothelial system, such as myelomatosis, leukemia and Hodgkin's disease, and conclude with the stimulating suggestion that amyloidosis may be a protein-storage reticulosis.

SUMMARY

1. A case of primary amyloidosis in a Bantu child is described.

2. The literature on primary amyloidosis is reviewed and emphasis laid on the absence of clear-cut anatomical distinctions between primary and secondary amyloidosis.

I should like to thank Dr. S. Selby for permission to publish this case and to express my appreciation to Dr. I. J. Grek and Dr. E. Basskind for their kind advice and criticism.

I wish to thank Dr. P. Keen and Dr. N. Feldman for the photographs.

REFERENCES

1. Bannick, E. G., Berkman, J. M. and Beaver, D. C. (1933): Arch. Int. Med., **51,** 978.
2. Bray, W. T. (1946): *Synopsis of Clinical Laboratory Methods,* 3rd ed. The C. V. Mosby Company.
3. Brown, H. A. and Selzer, G. (1944): Clin. Proc., **3,** 227.
4. Dahlin, D. C. (1949): Amer. J. Path., **25,** 105.
5. Dillon, J. A. and Evans, L. R. (1942): Ann. Int. Med., **17,** 722.
6. Dostrovsky, A. and Sagher, F. (1941): Arch. Dermat. Syph., **44,** 891.
7. Golden, A. (1945): Arch. Int. Med., **75,** 413.
8. Koletsky, S. and Stecher, R. M. (1939): Arch. Path., **27,** 267.
9. Lindsay, S. and Knorp, W. F. (1945): Arch. Path., **39,** 315.
10. Lindsay, S. (1946): Amer. Heart J., **32,** 419.
11. Lubarsch, O. (1929): Virschow's Arch. f. Path. Anat., **271,** 867.
12. Michelson, H. E. and Lynch, F. W. (1934): Arch. Dermat. Syph., **39,** 805.
13. Weber, F. P., Cade, S., Stott, A. W. and Pulvertaft, R. J. V. (1937): Quart. J. Med., **6,** 181.
14. Woolf, C. R. (1950): *S. Af. Med. J.,* **24,** 146.
15. Stemmerman, M. G. and Auerbach, O. (1944): Amer. J. Med. Sci., **208,** 305.
16. Wild, C. (1886): Beitr. z. path. Anat. v. z. allg. Path., **1,** 177. (*Quoted by* Lindsay, 1946.)
17. Wilks, S. (1856): Guy's Hosp. Rep., **2,** 105, Series 3. (*Quoted by* Dillon and Evans, 1942.)
18. Zemp, F. E. (1947): Ann. Int. Med., **26,** 448.

South African Medical Journal
Suid-Afrikaanse Tydskrif vir Geneeskunde

VAN DIE REDAKSIE

GENEESKUNDIGE BANKE

'N SLAGAARBANK

Vordering op die gebied van biologiese tegnieke het in die afgelope jare 'n groot verskeidenheid Geneeskundige Banke in die lewe geroep. By die lys wat Banke vir bloed, oë, bene, huid en saad insluit, moet nou 'n Slagaarbank gevoeg word.

Vyftien hospitale in die stad New York het onlangs 'n sentrale koöperatiewe Slagaarbank gestig wat waarskynlik die eerste van sy soort in die wêreld is. Dit is 'n bewaarplek waar are wat uit mense gehaal word nadat hulle gesterf het, bewaar word om op lewende persone oorgeënt te word. Die grootste aanwendingsveld is by die snykundige behandeling van aangebore hartkwaal, en aarweefsel van die Slagaarbank van New York is met welslae vir 'n volwassene gebruik. In hierdie geval is weefsel wat vir 24 dae bewaar is, aangewend om 'n deel van die hoofslagaar te vervang.

Slagaarweefsel vir die Bank moet binne vyf uur na oorlye verkry word en moet afkomstig wees van 'n taamlike jong persoon wat nie aan 'n ontaardingsiekte gely het nie. Om hierdie redes is dit klaarblyklik dat die voorraad weefsel verkry moet word deur middel van nadoodse operasies op persone wat skielik as gevolg van 'n ongeluk gesterf het; of van amputasies wat deur die ernstige besering van 'n ledemaat nodig gemaak is.

Die bloedvate wat gebêre word, word bewaar in voedende oplossings wat plasma, glukose, kiemdodende middels en soortgelyke stowwe bevat. Navorsing word gedoen in verband met metodes van ‚snelbevriesing' in die geval van mens-are sodat hulle vir 'n onbepaalde tyd bewaar kan word. Die snelbevriesingsproses het reeds geslaag geblyk in die geval van dier-are, wat vir 'n paar maande ongeskonde bewaar is.

Daar mag sekere wetsmoeilikhede in verband met die verkryging van materiaal vir 'n Slagaarbank wees. Dit is egter ondenkbaar dat daar enige moontlike hindernis kan wees wat die weg van hierdie belangrike reddingstegnieke ernstig versper.

BEENBANKE

Hierdie bewaarplekke waar gesonde bene (wat uit siek of beseerde persone gehaal word) vir ortopediese doeleindes of vir plastiese heelkunde bewaar word, word

EDITORIAL

MEDICAL BANKS

AN ARTERY BANK

Advances in biological techniques have created a great variety of Medical Banks in recent years. To the list which includes Blood Banks, Eye Banks, Bone Banks, Skin Banks and Semen Banks must now be added an Artery Bank.

Fifteen hospitals in New York City have recently established a central co-operative Artery Bank which is probably the first of its kind in the world. It is a repository where arteries taken from human beings after death are preserved for grafting into living persons. The greatest field of application is in the surgical treatment of congenital heart disease and artery tissue from the New York Artery Bank has been used successfully on an adult. In this case tissue stored for 24 days was employed to replace part of an aorta.

Arterial tissues for the Bank must be obtained within five hours after death and must come from a fairly young person who has not suffered from a degenerative disease. For these reasons it is obvious that the supply of such tissue must come from *post mortem* surgery of persons who have met sudden, accidental death; or from amputations necessitated by severe injury to a limb.

The stored blood vessels are preserved in nutrient solutions containing plasma, glucose, antibiotics and similar substances. Research is under way on methods of 'quick-freezing' human arteries so that they can be preserved indefinitely. The 'quick-freeze' process has already proved successful in animal arteries, which have been preserved intact for several months.

There may be certain legal difficulties about the acquisition of material for an Artery Bank. It is unthinkable, however, that any possible obstruction can seriously stand in the way of these important and life-saving techniques.

BONE BANKS

These repositories where healthy bones removed from sick or injured persons are preserved for orthopaedic or plastic purposes are maintained in a number of

in stand gehou by 'n aantal hospitale in die Verenigde State. Hierdie Banke waarmee slegs twee jaar gelede 'n aanvang gemaak is, het meegehelp om vele wat andersins lewenslank kreupel sou gewees het, se gesondheid te herstel. In hulle werking is hulle soortgelyk aan die Bloed- en Huidbanke wat reeds goed by die mediese gebruik van die Verenigde State gevestig is.

Bene word heel gebêre of word tot 'n growwe meel gemaal. In albei vorms word hulle in kiemvrye potte geplaas wat verseël en in vrieskaste gehou word. Teen hierdie lae temperature sal die bene vir 'n byna onbepaalde tyd hou. Hulle kan binne 'n paar minute ontdooi word.

Bene van hierdie Banke is gebruik by die ortopediese verbetering van gebreklikheid sowel as by plastiese heelkunde. Die meel wat van die bene gemaak word, is gebruik om holtes te vul. Die uitslae was baie geslaag.

Daar skyn aansienlike geleentheid in Suid-Afrika te wees om hierdie en ander Bankdienste te organiseer op 'n breë en gesonde grondslag wat noodsaaklike materiaal vir die pasiënt sowel as die snykundige maklik beskikbaar sal maak.

hospitals in the United States. Started only two years ago these Banks have helped to restore to health many who otherwise might have been crippled for life. They are similar in operation to the Blood and Skin Banks that are already well established in United States medical practice.

Bones are preserved intact or are ground into coarse meal. In either form they are placed in sterile jars, which are sealed and kept in 'deep-freeze' cabinets. At these low temperatures the bones will keep almost indefinitely. They can be thawed out in a few minutes.

Bones from these Banks have been used in the orthopaedic correction of deformities, as well as in plastic surgery. The bone meal has been used to fill cavities. Results have been very successful.

There seems considerable scope in South Africa to organize these and other Bank services on a broad and sound basis which will make vital materials readily available to the patient as well as the surgeon.

SIR REGINALD WATSON-JONES

DERDE RONDREISENDE ARTHUR SIMS-PROFESSOR

Op 29 September eersvolgende sal sir Reginald Watson-Jones van Londen na Johannesburg vertrek waar hy tot 9 Oktober 1950 sal vertoef. Sir Reginald besoek hierdie land as die derde van die rondreisende Arthur Sims-professore. Sy voorgangers was sir Hugh Cairns in 1948 en professor George Pickering in 1949.

Hy sal op 9 Oktober per vliegtuig van Johannesburg na Kaapstad vertrek en na vyf dae in die moederstad na Port Elizabeth (19 Oktober) en Durban (20 tot 22 Oktober) afreis en daarna sal hy na die Rhodesiës en aangrensende gebiede gaan. Hy sal op 14 November 1950 na Londen vanaf Kenia terugkeer.

Hierdie rondreisprofessorate het 'n waardevolle band tussen die universiteite en die mediese beroep van die Gemenebes ontwikkel. Sir Arthur Sims (wat verlede jaar tot ridder geslaan is en wat Nieu-Seelander van geboorte is met groot handelsbelange in Londen en Nieu-Seeland) is by die stigting van hierdie skema bygestaan deur die *Royal College of Surgeons* asook dié van *Physicians, Obstreticians* en *Gynaecologists*.

Van die bekleër van die professoraat word gewoonlik verwag dat hy Australië en Nieu-Seeland besoek. Vroeër in die jaar het sir Reginald afgereis om hierdie deel van sy toer *via* Kanada na te kom waar hy ongelukkig siek geword het. Hy moes na Engeland terugkeer sonder om sy reisplan te voltooi.

Sir Reginald besoek Suid-Afrika om die tweede deel van sy voorgenome reisplan te voltooi en ons is seker dat ons kollegas die geleentheid sal verwelkom om so 'n hoog aangeskrewe lid van die beroep te ontmoet en na hom te luister.

SIR REGINALD WATSON-JONES

THIRD ARTHUR SIMS TRAVELLING PROFESSOR

On 29 September next Sir Reginald Watson-Jones will leave London for Johannesburg where he will stay until 9 October 1950. Sir Reginald is visiting this country as the third of the Arthur Sims Travelling Professors, his predecessors having been Sir Hugh Cairns in 1948 and Professor George Pickering in 1949.

He will leave Johannesburg by air for Cape Town on 9 October and after five days in the Mother City will proceed to Port Elizabeth (19 October) and Durban (20 to 22 October) after which he will travel to the Rhodesias and adjacent territories. He will return to London from Kenya on 14 November 1950.

These travelling professorships have built up a valuable association between the Universities and the medical professions of the Commonwealth. In establishing the scheme, Sir Arthur Sims (who was knighted last year and who is a New Zealander by birth with large commercial interests in London and New Zealand) was assisted by the Royal Colleges of Surgeons, Physicians as well as of Obstetricians and Gynaecologists.

The incumbent is usually required to visit Australia and New Zealand. Earlier this year Sir Reginald started off to fulfil this part of his tour via Canada, where, however, he was unfortunately taken ill and had to return to England without completing his schedule.

Sir Reginald will be visiting South Africa with the intention of completing the second part of his planned itinerary and we are sure our colleagues will welcome the opportunity of meeting and listening to so eminent a member of the profession.

MIXED TUMOURS (BOTRYOID SARCOMATA) OF THE UTERUS

WITH THREE CASE REPORTS

G. P. CHARLEWOOD, F.R.C.S. (EDIN.), M.R.C.O.G.
Department of Gynaecology and Obstetrics, University of the Witwatersrand
AND
J. F. MURRAY, M.D.
Department of Pathology, South African Institute for Medical Research Johannesburg

Since Wagner [1] described the first case of botryoid sarcoma in 1854 some 99 cases in all have been described. (Glass and Goldsmith [2] considered that there were only 94 cases which could be reasonably included.)

Thiede [3] was the first to realize the mixed mesodermal nature of the tumour. Ehrlich [4] states that for a case to be completely acceptable it must contain embryonal myoblasts in combination with one or more heterologous mesodermal elements. He finds that there have been only 14 such cases. Murphy and du Shane [5] and Blumer and Edwards [6] described further cases which they claim were acceptable in this more restricted sense.

Site of Origin. These tumours are located either in the corpus or cervix uteri, there being a definite difference in the age incidence in the two localities.

Age Incidence. Meikle [7] shows that (very like carcinoma of the uterus) most mixed tumours of the corpus occur post-menopausally, the average age and maximum incidence being about 55, whereas the cervical tumours occur throughout menstrual life and also during infancy, the average age of the patients being 30 years.

Clinical Picture. Most patients present themselves with a fairly recent history of irregular vaginal bleeding and offensive discharge. On speculum examination the characteristic appearance is a mass of degenerating grape-like polypi. They are liable to be mistaken for fibroids or other simple polypi extruding from the cervix,[5] as occurred in the first case reported below. In practice the diagnosis is only made on histological examination, though if there are characteristic grape-like bodies bulging from the cervix the true nature of the tumour may be suspected, especially in very young nulliparous women, in whom other uterine growths are also rare. The importance of histological examination in every cervical polyp is obvious. In their later stages symptoms may be caused by pressure on, or invasion of neighbouring structures.

Prognosis. These tumours are extremely malignant with a special tendency to invade neighbouring organs. Metastases to lungs, pleura and bones occur in 20% of cases,[2] and take the form of spindle cells or myxosarcomata rather than the mixed structure of the primary growth.

The average duration of life after treatment is under one year, though von Franque [8] had one 10-year cure and Hartfall [9] one 5-year cure.

Duggan [10] believes that the presence of cartilage indicates extreme malignancy whereas the presence of fat indicates that the tumour is relatively benign.

Treatment. The usual treatment advised is total resection of uterus and adnexae, followed by deep X-ray therapy.

CASE 1

The patient, a Bantu woman aged 39, was admitted to the Coronation Hospital on 8 September 1949 (Case 6134C) complaining of something protruding from her vagina for three months, and contact bleeding for three months. Her menstruation had always been regular, lasting four days every month. She had had five normal full-time pregnancies, and one year before admission had a miscarriage at three months.

In recent months she had noticed a yellow, offensive, intermenstrual discharge. For the previous three months her husband had felt a vaginal mass during intercourse, after which there had been some bleeding. She had not noticed any loss of weight.

On general examination the patient appeared to be a healthy woman. Abdominal examination was negative.

On vaginal examination a large mass two and a half inches in diameter, soft in parts, was seen protruding from the cervix. It bled easily when touched. The uterus was not enlarged, and a bimanual examination was otherwise negative.

The clinical diagnosis was a submucous fibroid polyp, and the case was demonstrated to students as such.

At operation the tumour was found to have a thick pedicle originating from the endocervix. It was snipped through with curved scissors and sent for histological examination. There was no bleeding subsequently.

Unfortunately, before receipt of the histological report the patient discharged herself from hospital and could not be traced by the hospital social worker.

Histology. The specimen consists of a polypoid mass of fibrous tissue in which run numerous smooth muscle bundles. It is covered in part by chronic inflammatory granulation tissue, in part by stratified squamous epithelium and in part by a single layer of simple mucus-secreting epithelium of cervical type. Embedded in the stroma are numerous acinar spaces, the majority of which are lined by columnar epithelium of the type normally found in cervical glands (Fig. 1).

Occasional foci of loose myxomatous connective tissue resembling embryonic connective tissue are present in the subepithelial areas. In addition there are numerous cellular areas in the stroma composed of a mixture of spindle cells, myxoma cells and striated muscle cells. The muscle cells are also dispersed singly and in groups throughout the specimen (Fig. 2). In the main they show longitudinal and concentric striation but in a very few of them definite trans-

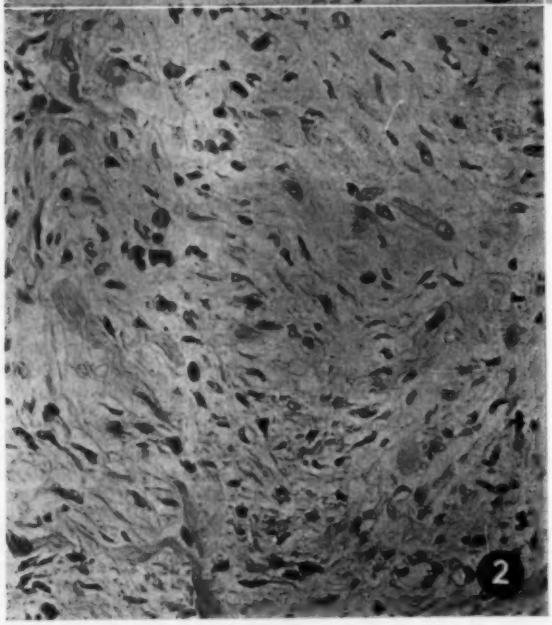


Fig. 1. Case 1. Acinar space embedded in the cellular connective tissue stroma and lined by tall mucus-secreting epithelial cells. A portion of cartilage is visible to one side. (H & E x 430. 6326E/49.)
Fig. 2. Case 1. Several striated muscle cells are visible in this area of the tumour. At this magnification only longitudinal striations are visible. (H & E x 430. 6326E/49.)
Fig. 3. Case 1. Striated muscle cells in which transverse as well as longitudinal striations are clearly visible. (Weigert's iron haematoxylin. x 1250. 6326E/49.)

verse striation is present (Fig. 3). The appearance of the spindle cells in the cellular areas suggests that they are relatively primitive cells capable of giving rise to a variety of cells including the striated muscle cells and the fibroblasts in the adjacent connective tissue and possibly to cartilage cells. Areas of definite simple cartilaginous tissue are embedded in the stroma and are surrounded by a condensation of fibrous tissue resembling a perichondrium (Fig. 4). Mitotic activity is present in the cellular areas of fibromyxomatous tissue but elsewhere is not observed. A diffuse scattering of plasma cells, round cells and phagocytes containing altered blood pigment is present throughout the whole tissue.

The patient reported back again on 26 March 1950 with a recurrence of vaginal bleeding. On vaginal examination: three polypoid, grape-like masses hung from the cervix. The uterus and cervix were, however, mobile. Her general condition was good. There were no changes seen on X-ray of the lungs. A Wertheim hysterectomy was performed (Fig. 5) but the growth appeared already to have passed far into the parametrium. She made a good immediate post-operative recovery. Sections of the polypoid mass and of the adjacent uterine wall showed histological features similar to those described in the original specimen.

Fig. 4. Case 1. Area of definite cartilage with surrounding spindle cells forming a perichondrium. (H & E x 430. 6326E/49.)

Fig. 5. Case 1. Macroscopic appearance of neoplasm growing from the endocervix when removed on 28 March 1950.

CASE 2

The patient, a European aged 56, was seen at Elim Hospital in May 1944. She had never had children but had one ectopic pregnancy in 1923 and two miscarriages subsequently. In 1933 a ventro-fixation operation was performed and in 1935 a sub-total hysterectomy.

On admission to hospital she complained of a bloody vaginal discharge after sexual intercourse. Vaginal examination revealed a small cauliflower growth hanging from the cervix. An amputation of the cervix was performed and the specimen sent for histological examination.

No further treatment was given and in a letter six months later her husband stated that the patient was well except that intercourse had to be discontinued altogether because of dyspareunia.

Histology. The specimen is a polypoid mass one inch in diameter. It consists of a stroma covered in part by a single layer of cuboidal epithelium, in part by tall mucus-secreting columnar epithelium and in one or two areas by stratified epithelium. Embedded in the stroma are acinar spaces lined by columnar epithelium of varying types, sometimes arranged as a single layer of cells and sometimes stratified. The main bulk of the stromal tissue consists of homogenous, eosinophilic fibrous tissue in which angiectatic blood vessels and relatively scanty fibrocytes are accompanied by a few plasma cells, lymphocytes and eosinophils. In some instances the fibrous tissue stroma runs right up to the columnar epithelium lining the acini, but around the majority of the acini (Fig. 6) and deep to the epithelial cells covering the surface of the polyp (Fig. 7) the fibrous tissue is replaced by a cellular loose connective tissue stroma which in many areas is myxomatous and bears a resemblance to embryonic connective tissue. This type of connective tissue is the distinctive feature of the growth and links it with the other cytologically more varied mixed tumours of uterine origin. No evidence of carcinoma and no cartilage, adipose tissue or muscle cells were found in the sections examined.

CASE 3

It was not possible to obtain clinical details of the third patient except that she was a European aged 41 complaining of vaginal bleeding and that she died a year after operation.

Histology. This specimen was received in such a poor state of preservation that the cytological detail was largely obscured. It can be observed, however, that there is a definite adenocarcinoma of the endometrium and that it is invading the perivascular lymphatics of the underlying myometrium (Fig. 8). The malignant epithelial cells are mainly arranged in irregular columns supported on their connective tissue stroma but here and there they show an imperfect acinar arrangement (Fig. 9). Embedded in the carcinoma are several foci of irregular atypical cartilage which are usually surrounded by a well-defined fibrous tissue layer (Fig. 10).

Here and there the cartilaginous tissue appears to be continuous with, and to merge into the carcinomatous tissue (Fig. 11). One or two foci having the appearance of adipose tissue have aso been observed (Fig. 12) but no striated muscle cells have been found in any of the sections examined.

DISCUSSION

Considerable confusion exists in the literature about how the endless variety of mixed tumours in the neighbourhood of the uterus and bladder base should be classified and named.

Cases 2 and 3 also seem to us to be correctly included as mixed tumours of the uterus, the first because of its characteristic embryonic type of connective tissue, and the second because of its islets of cartilage, both of which are heterologous structures characteristically found in these tumours; yet epithelial elements were prominently present in both. In Case 3 the epithelial tissue showed carcinomatous features.

The majority of authors, e.g. Murphy and du Shane,[5] pay little attention to epithelial elements within the

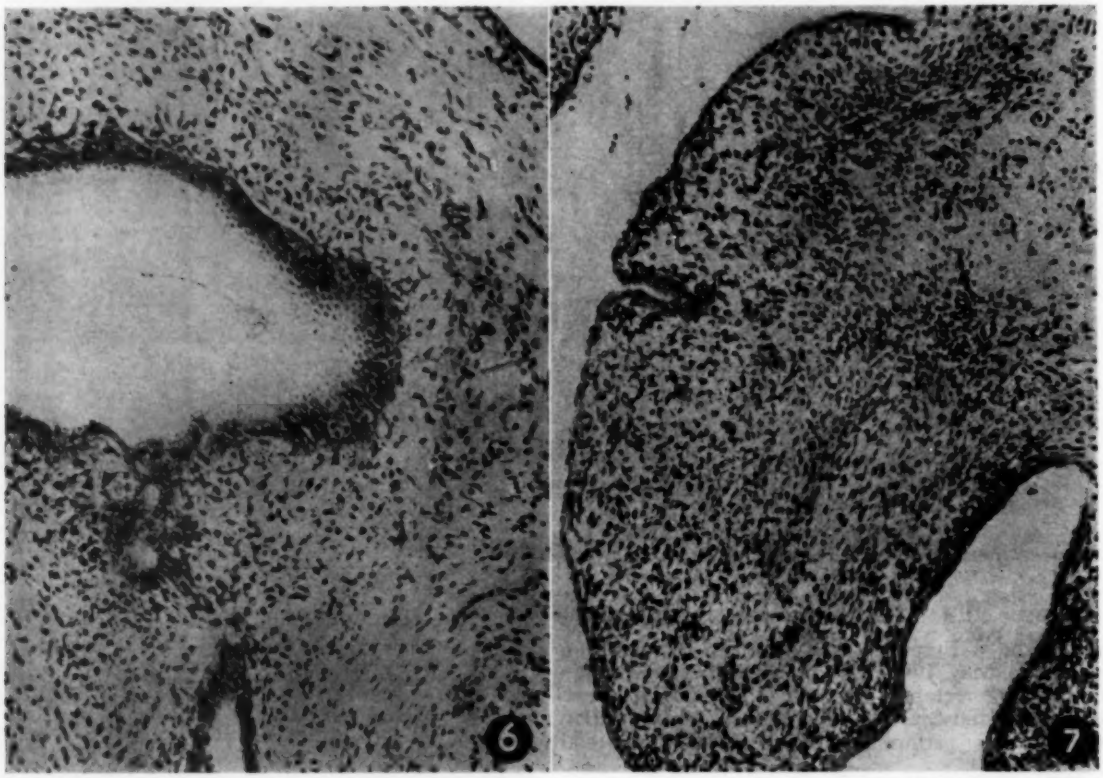
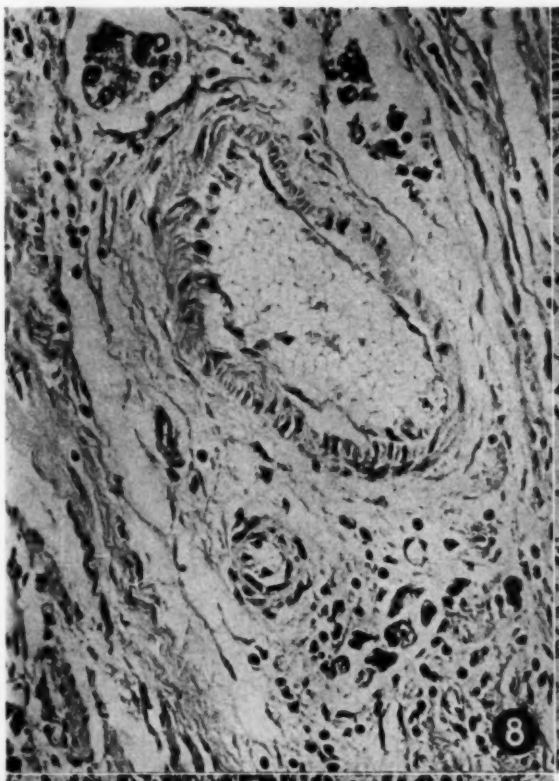


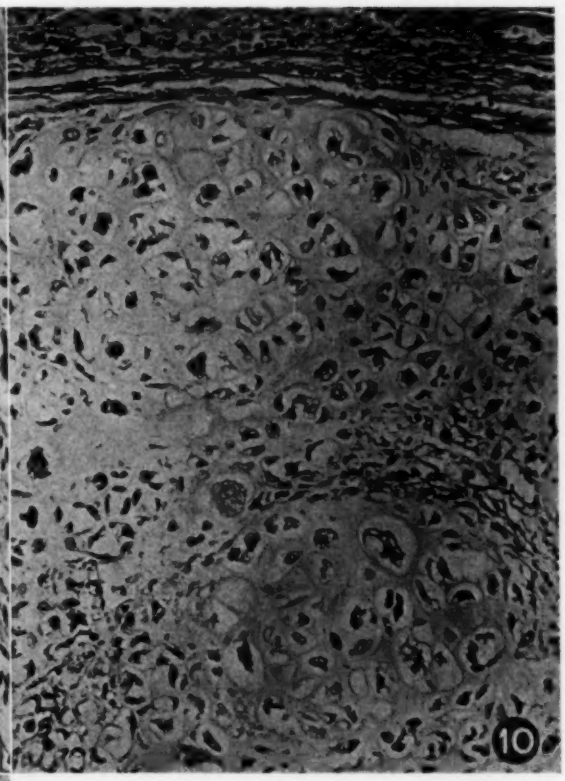
Fig. 6. Case 2. Loose myxomatous connective tissue stroma adjacent to acinar space. (H & E x 210. 1838C/44.)
Fig. 7. Case 2. Similar, slightly more cellular myxomatous connective tissue lying deep to the cuboidal epithelial cells which cover the surface of the polyp. (H & E x 210. 1838C/44.)

At the one extreme McFarland[11] groups together all mixed tumours of the urogenital region as 'dysontogenetic' tumours. At the other, Ehrlich[4] states that only uterine tumours in which embryonal myoblasts are present together with one or more heterologous mesodermal elements can be included as mixed mesodermal tumours of the uterus.

The first case reported here can be included in Ehrlich's exclusive group and is incidentally the first mixed tumour of the uterus to be reported in an African Bantu.

substance of the tumour, considering them to be merely downgrowths of normal epithelium. That this is at least not always the case is shown by such cases as numbers 2 and 3 above. Nicholson[12] shows that in his case there were several separate mesodermal mixed tumours over some of which the endometrium showed excessive physiological proliferation while over others it showed definite carcinomatous changes, a carcinosarcoma resulting. Sophian[13] reports a similar case and Meikle[7] quotes several others from the French and German literature. When Glass and Goldsmith[2]





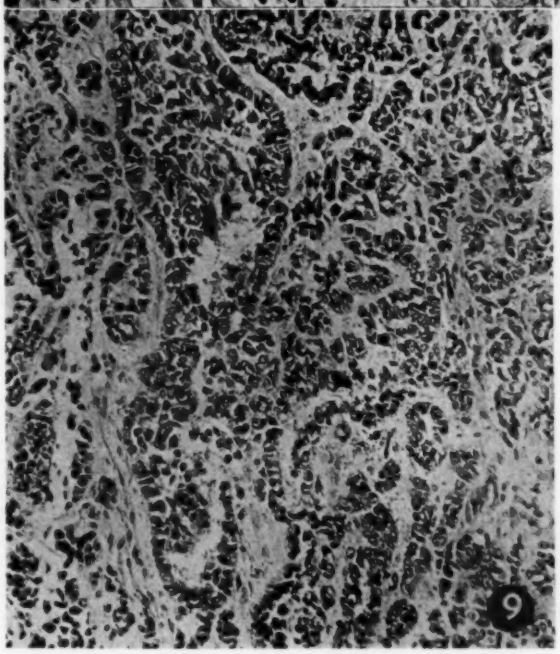


Fig. 8. Case 3. Tumour emboli in the lumen of the perivascular lymphatics in the myometrium. (H & E x 420. 10722A/37.)

Fig. 9. Case 3. An imperfect acinar arrangement of the neoplastic cells in one area of the tumour. (H & E x 195. 10722A/37.)

Fig. 10. Case 3. Irregular definite cartilaginous tissue bounded on one side by a layer of fibrous tissue in which some carcinoma cells can be observed. (H & E x 195. 10722A/37.)

surveyed the literature of mixed mesodermal tumours 13.9% of them were found to include epithelial elements.

SUMMARY

Three mixed tumours of the uterus are described. The first conforms with the rather unnecessarily restricted classification of Ehrlich. It is the first case to be described in an African Bantu.

The second contained embryonic-like connective tissue which links it with the wider group of uterine mixed tumours, though it contained a definite epithelial element.

The third case was mainly adeno-carcinomatous but islets of irregular cartilage indicate its relationship with the mixed tumours.

The importance of epithelial elements in uterine mixed tumours is stressed.

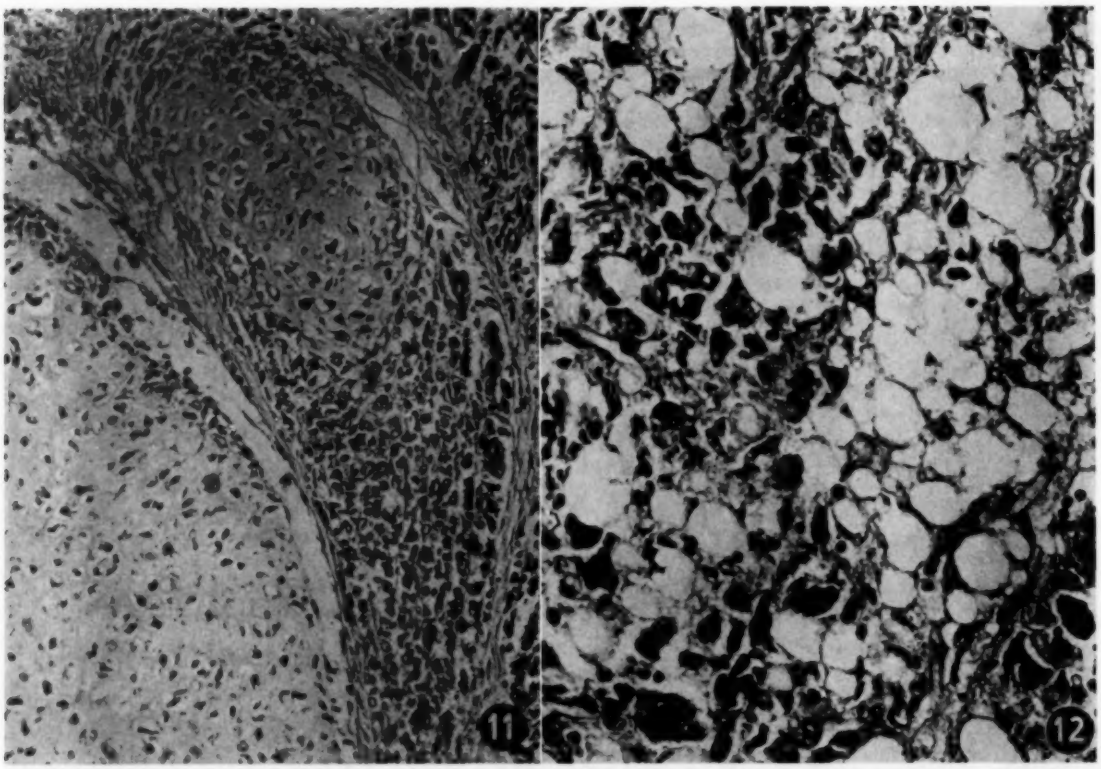


Fig. 11. Case 3. An area of cartilage in which the cartilage cells appear to merge into those of the adjacent carcinoma. (H & E x 195. 10722A/37.)
Fig. 12. Case 3. Focus having the appearance of adipose tissue embedded in which are numerous atypical hyperchromatic carcinoma cells. (H & E x 435. 10722A/37.)

We are grateful to Dr. Rosset of Elim Hospital for permission to use the case notes of Case 2.

We also wish to thank Mr. F. A. Brandt, M.A., B.Sc., of the South African Institute for Medical Research, by whom all the photo-micrographs were prepared.

REFERENCES

1. Wagner, E. *Quoted by* J. W. Williams (1894): Amer. J. Obstet. Dis. Wom., **29,** 721.
2. Glass, M. and Goldsmith, J. W. (1941): Amer. J. Obstet. Gynec., **41,** 309.
3. Thiede, M. (1877): Z. Geburtsh. Gynäk., **1,** 460. *Quoted by* G. J. Meikle.
4. Ehrlich, H. E. (1942): Amer. J. Obstet. Gynec., **43,** 1021.
5. Murphy, G. H. and Du Shane, J. W. (1948): Amer. J. Obstet. Gynec., **55,** 527.
6. Blumer, C. E. M. and Edwards, J. L. (1948): J. Obstet. Gynaec., **55,** 527.
7. Meikle, G. J. (1936): J. Obstet. Gynaec., **43,** 821.
8. von Franque, O. (1893): Z. Geburtsh. Gynäk., **40,** 183. *Quoted by* M. Glass and J. W. Goldsmith.
9. Hartfall, S. J. (1931): J. Obstet. Gynaec., **38,** 593.
10. Duggan, A. R. H. (1939): Med. J. Austral., **1,** 612.
11. McFarland, J. (1935): Surg. Gynec. Obstet., **61,** 42.
12. Nicholson, G. W. (1918): Guy's Hosp. Rep., **69,** 173.
13. Sophian, L. (1932): Amer. J. Obstet. Gynec., **24,** 911.

THE SIGNIFICANCE OF HAEMATURIA

P. J. DENNEHY, M.B., CH.B. (RAND), F.R.C.S. (ENG.)
Johannesburg

The disastrous aftermath when haematuria is regarded casually is well illustrated by the following case reports. These cases were dealt with during the short period of one year in a busy Johannesburg practice and form but a small cross-section of the end results of mishandled cases.

Case 1 (*Male aged 55 years*): Repeated attacks of haematuria extended over a period of three years. He was never investigated either by X-ray or cystoscopy. When first examined he had a large, infiltrating, inoperable growth of the right anterolateral wall of the bladder. He was treated by open cystotomy and fulguration. The prognosis is bad.

Case 2 (*Male aged 60 years*): Repeated attacks of

painless haematuria were treated by vitamin K, Sangostab, calcium and adrenalin. He was never investigated from a urological standpoint. Cystoscopy revealed a large malignant papilloma, obviously inoperable. Palliative resection with McCarthy's endoscopic loop was carried out. There is obviously a bad prognosis.

Case 3 (*Male aged 68 years*): He gave a long history of repeated attacks of haematuria which had been treated medicinally without investigation. Cystocopy showed a large, malignant, inoperable bladder growth. Palliative bilateral ureteric transplantation was performed. The patient died 48 hours after operation.

Case 4 (*Male aged 54 years*): In 1929 he had a bladder stone removed by suprapubic cystotomy. Following this he had, over a period of 12 years, five bouts of haematuria. This was never investigated and when first seen he had 12 oz. of residual urine and cystoscopy showed a large malignant growth of the bladder. Palliative bilateral transplantation was performed and to date the patient is doing very well.

DISCUSSION

The presence of blood in the urine is a real danger signal and to regard it casually is to court disaster. Its cause may be some minor trouble, the cure of which is a simple matter; but on the other hand it may be due to a serious condition which, if overlooked, may end in the patient's death after a period of extreme misery and suffering.

Blood does not occur in the urine of normal, healthy people and it is safe to assume that the majority of cases of haematuria, especially those unassociated with pain, are due to some lesion which, if uncorrected, will cause loss of health and eventually life itself. Such being the case, haemorrhage is the one urinary symptom calling for immediate and urgent investigation with a view to determining its source and cause. To depend on medical treatment combined with rest in bed is often a shot in the dark, the old hit or miss philosophy, and amounts to extremely culpable and dangerous neglect.

Importance of a Clinical History. By a competent investigation of the clinical history, combined with a thorough examination, it is often possible to arrive at a provisional diagnosis which, in a large number of cases, is substantiated. If blood has appeared suddenly, painlessly and profusely, it is reasonable to assume that the most likely cause is a villous papilloma of the bladder, or a tumour of the kidney. However, in cases of hypertension profuse, painless haematuria is not uncommon. If bleeding occurs apart from urination, it is urethral in origin, but this may be an early symptom of carcinoma of the prostate. It is important to determine if bleeding occurs throughout the whole act of urination or is merely terminal. If it occurs when the bladder base is being compressed, it is due frequently to a localized inflammation of that part (a trigonitis) or comes from the congested surface of a prostate which is the site of an intra-vesical protrusion. Terminal haematuria, apart from bilharzia, may also be due to vesical tumour, ulcer or stone. If the urine contains pencil-like clots, it is likely, though not certain, that the bleeding is of renal origin. Bleeding associated with suprapubic pain or perineal discomfort may be due to a prostatitis or perhaps a malignant growth.

The association of renal colic with haematuria often indicates calculus, though colic in association with hypernephroma or tuberculosis of the kidneys is not uncommon. If in the presence of painless haematuria colic occurs, the latter phenomenon is due, probably, to a blood clot in the renal pelvis or ureter.

Before the advent of the cystoscope, one had to rely on one's clinical sense and the symptoms of the patient to determine the cause of the bleeding. Thanks to the studies which the cystoscope makes possible, we can now get an answer of sufficient certainty to avoid great risk to the patient. Those who deny the patient with haematuria this very valuable means of diagnosis will often have much to regret.

Though from the character of the blood-stained urine one can get a rough idea where the haemorrhage is taking place, too much reliance on this has lost many lives. It was thought once that 'coffee ground' urine was due to renal bleeding, but this has been found to be fallacious because the 'coffee ground' appearance depends on the reaction of the urine and how long the blood has been in it. Thus 'coffee ground' urine can come from any part of the renal tract proximal to the membranous urethra. Urine with bright red blood was thought to come from the bladder, yet with almost equal frequency it comes from the kidney.

Thus we see that the reliance on a symptomatic diagnosis of haematuria is a great injustice to the patient, for though one may thus diagnose the source of the bleeding, it is far more important to discover its cause.

Provided there are no contra-indications to cystoscopy, the patient with haematuria should be cystoscoped while he is bleeding. This makes the diagnosis of the source of the blood a relatively easy matter and may obviate much needless investigation. In the presence of profuse haemorrhage in a patient with prostatic enlargement, known tumour or stone, it is often better judgment to check the bleeding by putting the bladder at rest by means of continuous urethral drainage before resorting to cystoscopic studies.

Though there are systemic conditions which manifest themselves in haematuria such as haemophilia, purpura and leukaemia, yet even in these it is not safe to overlook the possibility of co-existing true urogenital pathology.

Physical Examination. After a careful study of the clinical history, the physical examination is proceeded with and may yield further valuable information about the source of the bleeding. If one kidney is palpable it may mean tumour or other disease that causes bleeding; but, on the other hand, the enlarged kidney may be healthy, the site of compensatory hypertrophy, whereas it is the other kidney that is diseased. The commonest cause of bilateral enlargement is congenital polycystic disease. In this condition the amount of renal tissue is markedly diminished and the reserve dangerously low, and any hasty exploratory operation may be followed by fatal consequences.

Ancillary Methods. The examination of the

centrifuged urine for malignant cells is seldom successful. However, occasionally a piece of villous papilloma may be found in the urine and this finding is most helpful.

Although in most urological disorders it is wise to perform a plain X-ray of the entire urogenital tract, this does not always indicate the source of the urinary bleeding.

Intravenous pyelography should always be carried out and in a proportion of cases this will clinch the diagnosis. In some cases retrograde pyelograms will have to be performed. Even when the diagnosis of vesical papilloma has been made cystoscopically, an intravenous pyelogram should be performed to exclude the possibility of seedling growths from a papilloma higher in the renal tract. A rectal examination should always be performed, for by it the state of the prostate can be determined, a stone may be palpable either in the bladder or in the lower end of the ureter, or extravesical induration might suggest advanced malignancy.

The true origin of the bleeding can be recognized usually by using the cystoscope. There is seldom need for a general anaesthetic and Cocaine Hydrochloride 0.25 gr. in 4 drachms of 0.5% Sodii Bicarb. gives excellent anaesthesia and is not associated with any danger in spite of many current views on the subject of cocaine as a urethral anaesthetic.

CONCLUSION

1. Haematuria must be looked upon as a real danger signal and every case must be regarded as potentially serious. This may necessitate a somewhat tedious examination, but the ultimate correct diagnosis is in most cases certain.

2. There is no place for symptomatic treatment in cases of haematuria. Cystoscopy can make a great contribution to diagnostic exactitude and should be employed in every case of haematuria without exception.

I wish to express my thanks to Dr. R. Campbell Begg for permission to publish this paper.

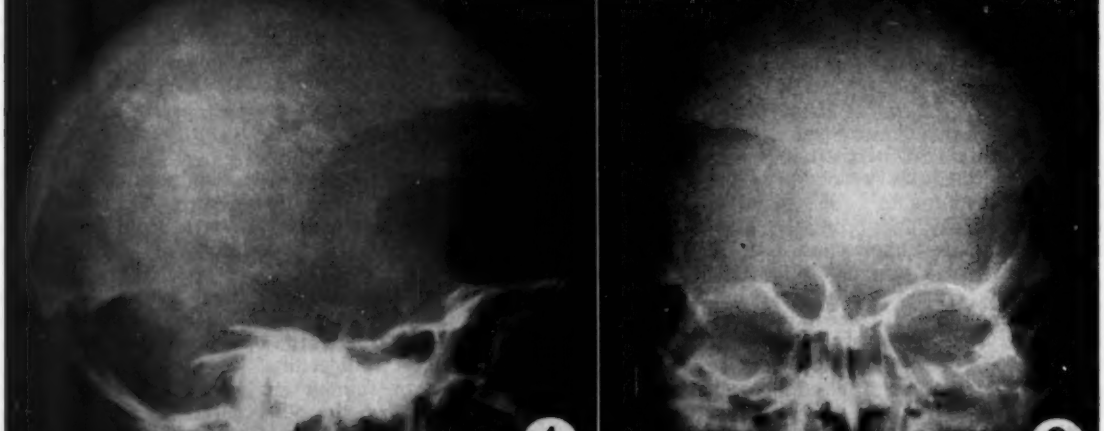
RADIOLOGICAL CASE BOOK. XXX

OSTEOPOROSIS CIRCUMSCRIPTA (PAGET'S DISEASE)

C. J. B. MULLER, M.B., CH.B., D.M.R.
Cape Town

One of the interesting early manifestations of a disease that can be detected skiagraphically but not clinically is osteoporosis circumscripta of the skull in Paget's disease.

Osteoporosis circumscripta has a characteristic and striking skiagraphic appearance, viz., an extensive



The correlation of the former with the latter is an excellent example of the medical progress due to the comparison of the radiological with the pathological findings. This is admirably described in an article by Kasabach and Gutman.[1]

osteoporosis that may be localized, usually in the frontal or occupital area; or may extend across the base of the vault in a broad band. It has a scolloped

margin and shows an abrupt transition from normal to osteoporotic bone. The bone structure is not destroyed as is metastases, myeloma and xanthomatosis, from which it must be differentiated. The vagueness of the symptoms, headache and indefinite pain in the neck, and the absence of any signs, makes a clinical diagnosis difficult. Biochemical changes do not occur until the generalized changes of Paget's disease take place.

Up to 1937, of 47 cases in the literature, 32 were associated with Paget's disease elsewhere in the skeleton. In five cases that initially showed no changes elsewhere, Paget's disease in other bones was detected within two to eight years. It may precede the other bony changes of Paget's diseases by as much as 20 years.

Case History. A white South African woman, aged 50 years, complained of vague headache and pains in the neck and shoulders. Clinically cervical osteoarthritis was suspected. A complete systemic clinical examination was negative for pathology. X-ray films of the cervical region revealed normal vertebrae but a portion of the skull, seen on the films, was obviously abnormal and further views showed a broad band of osteoporosis extending across the base of the vault typical of osteoporosis circumscripta (Figs. 1 and 2). Skiagrams of the limbs, the spine and pelvis showed no abnormality. The serum chemistry and blood examination were normal.

REFERENCE

1. Kasabach, Haig H. and Gutman, Alexander, B. (1937): Amer. J. Roentgenol. Rad. Ther., **37**, 577.

MEDICO-LEGAL SECTION

TESTAMENTARY CAPACITY : APHASIA

LEWIN *VERSUS* LEWIN*

(TRANSVAAL PROVINCIAL DIVISION)

1949. *May* 3-6, 9-13, 16-20, 23, 25, 27, 30; *June* 1, 2, 3, 6; *August* 2. Roper, J.

Impairment of mind resulting from aphasia is in the legal aspect in no way different from the mental enfeeblement resulting from illness, disease or old age.

In determining whether or not a testator had the necessary testamentary power it is not sufficient that he understood or intended the dispositions which he was making in his will: it is necessary further that he should have been able to comprehend and appreciate the claims of his various relations upon his bounty, without any poisoning of his affections, or perversion of his sense of right, due to mental disorder, and generally he should have had the ability clearly to discern and discreetly to judge of all these things and all those circumstances which enter into the nature of a rational, fair and just testament.

Plaintiff, the surviving spouse of the testator, sought to declare invalid a will executed by the testator on 15 April 1948, which made a radical departure from the terms of a previous will, on the grounds (*a*) that the Buerger's disease from which the deceased suffered had existed not merely in his limbs but in his brain and had so impaired his judgment and discretion that he would have been incapable of making a will even if he had not suffered from a stroke; (*b*) that the aphasic condition to which he was reduced by the stroke on 25 November 1944, had been such as to deprive him of testamentary capacity and (*c*) that on the medical evidence the special precautions which are necessary to ensure that the real wishes of aphasics are truly recorded had not been adopted. The medical evidence showed that aphasia from which the deceased was suffering was of the combined motor and sensory type, i.e., it indicated that the brain lesion caused by the thrombosis had involved not only the parts of the brain which subserve the function of speech, but those which govern the comprehension of ideas, the understanding of written and spoken language and the ability to convey ideas to others. At the time of the execution of the will the testator was under curatorship by order of Court.

Held, as to (*a*), that there was no evidence which convinced the Court that the changes, which Buerger's disease is alleged to bring about in a personality, were sufficiently grave to have impaired the testator's capacity or to have entered into his making of the disputed will.

Held, as to (*b*), though the deceased had understood and intended the main provisions of his will, that they were different from the provisions which he would have been likely to have made had it not been for his brain lesion and his resulting aphasic condition, and the difference was due to a perversion of his mental balance, judgment and affections caused by that lesion.

Held, accordingly, without dealing with ground (*c*), that judgment should be granted in plaintiff's favour.

Held, further, as the will was invalid, that the defendant was not entitled to the executorship to which he had been appointed and was not entitled to be paid his attorney and client costs out of the estate.

The question whether the *onus* was on the defendant because (1) at the time of the execution of the will the testator was under curatorship and (2) of the proof that in the early stages after the deceased's stroke he was clearly without testamentary capacity raised but not decided.

Roper, J.: The plaintiff is the widow of the late Meyer Maxwell Lewin, and she sues, in her personal capacity and as the mother and natural guardian of her two minor children, for an order declaring null and void, on the ground of want of testamentary capacity, a document purporting to be the last will and testament of the deceased and executed by him on 15 April 1948. The deceased was in his lifetime a medical practitioner, and practised for some years at Salisbury in Southern Rhodesia. The marriage between him and the plaintiff took place in 1927 and the two children of the marriage were born in 1928 and 1931. The history of the marriage in so far as it bears upon the present case may be said to begin in the year 1933.

In that year, while the deceased was in the enjoy-

* The appeal which had been noted was not proceeded with. —*Ed., S.A. Law Reports.*

Reprinted with additions by permission of the publishers (Messrs. J. C. Juta and Co.) from the *S.A. Law Reports*, November 1949 (4).

ment of a lucrative practice at Salisbury, he was suddenly obliged to undergo an amputation of his left leg below the knee. The cause of this operation was not definitely proved in evidence in the present case but there appears to be little doubt that it was the complaint known as Buerger's disease. After his recovery from this operation he resumed practice in Salisbury, and in 1937 built a double-storeyed building, named 'Leander House', comprising on the ground floor his surgery, and other rooms required for his medical practice, and on the upper floor six furnished rooms and a flat which were let to tenants. In October 1938, he contracted phlebitis in the leg and in December 1938, pulmonary emboli, as a result of which he was admitted to hospital and, according to the plaintiff, she was advised that the deceased would not be able to continue his practice. In the beginning of 1939, on his leaving hospital, the deceased decided to go overseas for treatment. Before doing so he executed a will, dated 13 February 1939, in which, after bequeathing to his two children the proceeds of certain two life insurance policies, he formed the residue of his estate into a trust, the income whereof was to be paid to the plaintiff during her lifetime, provided that in case of her remarriage she was to receive only one-third of the income, the remaining two-thirds going to the two children. On her death the *corpus* of the trust was to be divided between the children or their issue by representation.

While he was overseas the war broke out, he joined the Royal Army Medical Corps, and was sent to the Middle East, and subsequently to Basutoland, apparently as medical officer to the African Auxiliary Pioneer Corps at Maseru. In December 1942 he was boarded out of the army as medically unfit, and applied for a pension but was unsuccessful. It appears from his application for the pension that among the causes of his breakdown in health were pains and swellings in the leg in which he had previously had phlebitis, with deterioration of the circulation. He had also had pulmonary emboli and a right subclavian thrombosis while overseas.

On his discharge from the army he acquired a farm near Westminster in the Orange Free State, where he lived with his family, combining farming operations with a country medical practice, until August 1944. He then developed pains in the arms and in the right leg, in consequence of which he was removed to the Groote Schuur Hospital and underwent a lumbar sympathectomy for Buerger's disease on 10 November 1944. On 28 November 1944, while still in the hospital he developed a stroke, which resulted in paralysis of the right side of the body and aphasia. Thereafter he lost by amputation first the toe of his right foot and then the right leg. He remained in Groote Schuur Hospital until August 1945. During this period the plaintiff sold the Westminster farm and the movable assets upon it, including the deceased's medical instruments. After some time occupied in settling his affairs she moved to Cape Town, obtained accommodation near the Hospital, and was with the deceased for the last five months of his stay there.

When he was sufficiently recovered to be removed from the hospital the plaintiff took the deceased to the home at Oudtshoorn of the deceased's brother and sister-in-law, Mr. and Mrs. I. B. Lewin, where they stayed until December 1945. Thereafter they spent a month or two in a house at Mossel Bay with Mrs. I. B. Lewin and about six weeks in a boarding-house in the same place, whereafter they proceeded to East London, the home of the plaintiff's parents. At East London accommodation was found for the deceased in a nursing home, while the plaintiff lived with her brother and sister-in-law, obtaining employment in the town and visiting the deceased daily. In the course of time the deceased became dissatisfied with the nursing home and expressed a wish to return to Oudtshoorn. It was not possible, however, for the I. B. Lewins to accommodate him, and as the result of a suggestion by Mr. I. B. Lewin it was arranged that the deceased should be taken to Cape Town with a view to his entry into the Jewish Aged Home which was then nearing completion. The deceased accordingly left East London towards the end of 1946, while the plaintiff remained at East London and continued in the employment which she had secured there.

During the period of his stay in East London the deceased had developed extravagant habits, and a suggestion was made, and discussed between the plaintiff, her attorney Mr. J. A. Shapiro, and Mr. I. B. Lewin, that he should be placed under curatorship on the ground that he was mentally incapable of managing his affairs. According to Shapiro this suggestion was dropped at the instance of Mr. I. B. Lewin because of the slur which it would have cast upon the family. During this period also the deceased developed a dislike of the plaintiff, described by some of the witnesses as a 'hatred' of her, and seems to have conceived the idea of a divorce from her. There is also some evidence of a dislike by him of the children at this stage.

After leaving East London, the deceased was for a short period at Hermanus while waiting for the Jewish Aged Home to be opened, and was eventually admitted into the Home in November 1946. Owing to his extravagance while in this institution the suggestion of a curatorship was revived, and eventually, with his consent, on the ground of physical incapacity, he was placed under the curatorship of a Cape Town accountant named Goldberg, by order of the Cape Provincial Division of the Supreme Court dated 2 July 1947.

Towards the end of July 1947 there was a deterioration in the health of the deceased and the plaintiff was sent for from East London where she was still in employment. Having arrived at Cape Town she secured accommodation near the Jewish Home in order to be in a position to visit the deceased daily, gave up her employment at East London and secured other employment in Cape Town, in order to be near the deceased and to assist towards the recovery of his health. During this period the evidence shows that her relations with the deceased were harmonious and happy.

By October 1947, the health of the deceased had improved, and on the 10th of that month he was taken by the plaintiff to 'Avalon', an institution for disabled ex-servicemen established by M. de B. Nesbitt,

the author of *The Road to Avalon*, among the mountains of the Tulbagh district, and described as a self-rehabilitation centre. There he was left while the plaintiff returned to her employment in Cape Town. Relations between the two spouses were still harmonious at this stage. The plaintiff paid two further visits to Avalon, the first of which was without incident. At the second visit, however, which was towards the end of November 1947, an occurrence took place which was to have unfortunate results upon the relations between the spouses. Immediately after the plaintiff's arrival in the early afternoon, the deceased expressed a desire to have sexual intercourse with her, but she was reluctant to agree. This led to an angry scene which resulted in her running in a distressed condition to the residence of the Nesbitts, where she slept that night. On the following day she tried to reason with the deceased, but found that he would have nothing to do with her. On her leaving that day to return to Cape Town he told her to go and not to return. She never revisited him, this apparently on Nesbitt's advice. She wrote to him thereafter but received no reply to her letters. There is clear evidence that immediately after this incident the idea of a divorce from the plaintiff revived in the mind of the deceased.

The deceased remained at Avalon until the end of February 1948. He had before that become dissatisfied with his existence there, and Nesbitt had suggested that he should be removed. On the return of his son Gerald from a trip overseas the latter was sent by the plaintiff to fetch him, and Gerald took him to Johannesburg by car. Here there was difficulty in finding accommodation for him, and eventually the son took him to the defendant, who is a chemist and a nephew of the deceased, at the place of business of the latter in Nigel. The defendant agreed that the deceased should live with him and his wife at a hotel in Nigel where they were residing, and accommodation was arranged accordingly. With the exception of a period spent in hospital, the deceased lived thereafter with the defendant and his wife, either in the hotel referred to or in a house subsequently rented by defendant, until his death upon 7 May 1948.

After he had lived in the hotel for some 12 days the deceased developed severe abdominal pains which were diagnosed by a local practitioner as due to appendicitis. He was removed to the Nigel Hospital and operated upon on 14 March. Before the operation he expressed a desire to make a will and with the assistance of the doctors and nurses one was executed by him which was subsequently destroyed. On his discharge from hospital he expressed a wish to see an attorney and in due course executed a further will on 15 April 1948. This is the document which is the subject of dispute in the present case. In it the deceased made the following main provisions. He directed that all debts due by him should be paid, including an amount of £700 due to one E. S. Myers of Boksburg. He directed that his executor should give effect to the conditions of his antenuptial contract whereunder he ceded and made over to his wife the proceeds of a life insurance policy for £1,000. To his nephew, the defendant, he bequeathed 'Leander House' subject to any mortgage bond that might be on the property at the date of his death. To his son Gerald he bequeathed his motor-car and £1,000, and the residue of his estate was left to the two children in equal shares.

This will therefore made a radical departure from the terms of the will of 1939. The wife was deprived of all the benefits conferred upon her in the earlier document, while the inheritances of the two children were substantially reduced by the bequest of 'Leander House' to the defendant. Incidentally I may point out that in the antenuptial contract the deceased had settled upon the plaintiff not one policy for £1,000, but three such policies, and furniture to the value of £600.

The validity of this will is attacked by the plaintiff on three main grounds. It is said that on the evidence the Buerger's disease from which the deceased suffered had existed not merely in his limbs but in his brain and had so impaired his judgment and discretion that he would have been incapable of making a will even if he had not suffered from a stroke; it is contended further that the aphasic condition to which he was reduced by the stroke on 25 November 1944, was such as to deprive him of testamentary capacity. The third ground is based upon certain medical evidence which was put before the Court as to the difficulty of ascertaining the real wishes of aphasics, and as to the consequent necessity of special precautions in order to ensure that their testamentary wishes should be truly recorded. It is argued that as the evidence shows that no such precautions were taken it is not established that the will embodied the testamentary wishes of the deceased.

I shall deal briefly with the first of these contentions. Buerger's disease, or *thrombo-angiitis obliterans*, consists of an inflamed condition of the arteries which produces pain in the parts affected and in due course results in occlusion of and thrombosis in the diseased artery, and the decay of the parts of the body served by it. This is often manifested by gangrene in the extremity of an affected limb. Medical science has established that the disease may exist in the arteries of the brain as well as those of the limbs and body. In such a case, it is said, the patient undergoes a subtle change in his personality resulting in grandiosity, extravagance, euphoria, and a consequent impairment in his powers of normal judgment and discretion. Evidence was led in the present case to show that before the stroke there were such changes in the personality of the deceased, dating from the time of his first amputation, that namely which took place in 1933; and it appears to me quite probable that these were due to the Buerger's disease which had obtained a hold upon his body. There was no evidence, however, which convinced me that the changes in his personality were sufficiently grave to impair his testamentary capacity or to have entered into his making of the disputed will. If his testamentary capacity was impaired at the time of the execution of that document, the cause must in my view be sought in the condition resulting from the stroke of November 1944. The substantial ground of attack upon the will is therefore the second.

In considering the effect of the stroke upon the mental state of the deceased it is necessary first to ascertain so far as possible its nature and extent.

The medical evidence establishes that the stroke was due to injury of the main middle cerebral artery on the left side, which affected the frontal and temporal areas of the brain. There was some discussion during the evidence as to whether the injury was caused by a thrombosis resulting from arterial disease in the brain, or by an embolus due to some affection of the heart. Drs. Casserley and Suzman, both of whom saw the deceased, and the former of whom had actually watched him during the progress of the stroke, formed the opinion that it was due to the former of the two causes. Dr. Watt, who was called for the defence, but had never seen the deceased, was inclined to put forward the theory that it might have been due to an embolism; but in cross-examination he admitted that on the evidence the probabilities were somewhat in favour of a thrombosis. I accept the view of Drs. Casserley and Suzman on this point.

A difference of opinion among the medical witnesses also revealed itself on the point whether, assuming that the injury was due to thrombosis, the arterial disease which led to it was the Buerger's disease which had already manifested itself in other parts of the patient's body, or arteriosclerosis. Drs. Casserley and Suzman were inclined to the former view, while Dr. Watt, relying mainly upon the absence of evidence of certain premonitory symptoms usually associated with Buerger's disease in the brain, was inclined to the latter opinion. It is not necessary for me to come to a finding upon this point in view of the expert evidence that either cause could result in the same degree of impairment of the intellectual faculties of the patient. There is a suggestion in one passage of Dr. Watt's evidence that the possible degree of ultimate recovery would be less in the case of Buerger's disease than in that of arteriosclerosis, but on the whole the evidence is that there is no important difference in result between the two conditions. There was also considerable debate during the hearing as to whether the aphasia of the deceased which followed the stroke was purely motor, affecting only his powers of speech, or a combined motor and sensory aphasia which affected not only his speech but also his powers of comprehension. I do not propose to discuss the evidence on this point, as in my opinion it was overwhelmingly in favour of the view that the aphasia was of the latter type. This indicates that the brain lesion caused by the thrombosis had involved not only the parts of the brain which subserve the function of speech, but those which govern the comprehension of ideas, the understanding of written and spoken language, and the ability to convey ideas to others; as it was put by Dr. Casserley, it affected the ability to ' proposition ', that is, to indulge in consecutive logical thought and the transference of thought and ideas. The general deterioration of the brain, more particularly if the frontal areas are affected, would, according to Dr. Suzman, affect his power of exercising judgment and discretion and discrimination. The evidence of Drs. Casserley, Suzman and Geerling, which I accept, and which was not disputed upon this point by Dr. Watt, was that a very considerable area of the deceased's brain on the left side was affected as a result of the thrombosis and that the damage done to it was extensive.

Dr. Casserley, who saw the deceased almost daily for the first three or four months of his stay in Groote Schuur Hospital, and thereafter at intervals until he left the hospital, described in detail the mental condition of the patient during this period. His evidence leaves no doubt that throughout the period, although there was some slight degree of recovery after the initial shock of the stroke, the aphasic condition of the deceased was so severe that it would have been impossible for him to have made a will except, perhaps, one of the very simplest description. There was a failure of comprehension; great difficulty on the part of the deceased in understanding spoken, and complete inability to understand written, language; great difficulty on the part of the hearer in understanding what the deceased intended to convey and uncertainty as to whether he was correctly understood; and inability in the deceased to exercise normal judgment or discretion in relation to his affairs. I may say that I accept the evidence of Dr. Casserley as to the condition of the deceased during this period without qualification. This witness impressed me as being completely impartial and detached; owing to the fact that the deceased was a medical practitioner he took an especial interest in his case, and his observations of the mental and physical condition of the patient appeared to me to have been both careful and well informed. Dr. Watt agreed that during this period, according to the evidence, the deceased was in a state of almost global aphasia, with impairment of judgment, and that, during at any rate the first three months, it was very doubtful whether he could have understood even a simple will. The question is, therefore, whether such an improvement had occurred at the time of execution of the disputed will, as would have enabled the deceased to regain his testamentary capacity.

The medical evidence shows that a thrombosis occurring in a cerebral artery stops the flow of blood to those parts of the brain which are served by the affected artery, and by so doing causes the atrophy and death of all those parts which lie beyond the point at which the stoppage occurs. Such atrophy occurs within a comparatively short period, and when once it has taken place it is irreversible. The part so affected can never recover, at any rate completely. The adjacent blood vessels in the healthy tissues surrounding the affected part, however, send fresh, small blood vessels into it and so restore it to some extent, and there is a gradual assumption by other parts of the brain of the functions formerly carried out by the affected part. This results in a certain degree of recovery of the intellectual powers of the patient. Where there is disease of the arteries (such as arteriosclerosis or Buerger's disease) in old and debilitated subjects the degree of recovery is usually less than where there is no such disease, as in the case of young subjects.

There is evidence that the deceased had to some extent recovered his mental powers by the date of execution of the disputed will, and I will deal with the extent of that recovery at a later stage.

In addition to the deterioration of the purely mental powers of a patient, medical science shows that aphasia resulting from a thrombosis such as occurred in the case of the deceased is accompanied by marked psycho-

logical changes. The following extracts are taken from an article on *Aphasia and Will Making* by Byron Bramwell, which is stated by Dr. Geerling to be authoritative, and from which no substantial dissent was expressed by any medical witness:

'In many cases the emotional faculties are markedly disturbed; and in other cases, as a result of the brain disturbance (the brain lesion upon which the aphasia depends or with which it is associated) the judgment and affections of the patient are perverted, so that he takes unreasonable likes and dislikes to relatives, friends, persons who are in contact with him, etc.'

Again:

'It must be remembered, as I have more than once pointed out, that in many cases of aphasia the cerebral lesion not only disturbs the speech functions or destroys the speech centres, but produces more or less marked and widespread deterioration of the mental and intellectual faculties. In many cases of aphasia and hemiplegia the emotional faculties are distinctly altered; there is often impairment of cerebral and emotional control; many aphasic patients laugh and cry too easily; they are irritable, they get angry or excited about trifles, they take likes or dislikes to those who are in contact with them without obvious or sufficient cause. In other cases, as I have already pointed out, the memory, reasoning power, judgment, and in fact all the intellectual faculties are more or less, and in some cases, profoundly disturbed... In many cases the powers of attention and mental concentration are materially impaired. In many cases the brain power is easily exhausted, and the patient who at the commencement of the conversation or examination is mentally clear and alert may, in the course of a short time, become so mentally confused or dull that he is unable to understand what is said to him or to express himself so that he could make himself intelligently understood. In other cases the will is enfeebled; some aphasic patients assent to propositions or proposed lines of action to which they would not have assented before the onset of the aphasia; or they fail to dissent, or only feebly dissent, from other propositions or proposed lines of action from which they would have originally dissented before the onset of the aphasia.'

And again:

'Even if the wishes of the patient have been satisfactorily elicited and understood, even if the physician is satisfied that the will as a whole and its individual clauses have been clearly understood and comprehended by the patient—after, in short, a will in accordance with the wishes and desires of the patient has been correctly executed—a further question may arise—and it is often a most difficult question to determine—whether, in consequence of the brain lesion with which the aphasia is associated and on which the aphasic symptoms depend, the patient's mental balance, judgment and affections have been so perverted that he has taken an unreasonable liking or disliking for certain persons whom he has included in, or excluded from, his will. In those cases in which the provisions of the will, whether positive or negative, are peculiar or altogether unexpected, in other words are, so far as can be judged of, different from the provisions which the patient would have been likely to make when in a condition of brain health (i.e. before the onset of the cerebral lesion and the aphasic condition), the question naturally arises whether such provisions are the result of a disordered mental condition due to disease, i.e. are the result of the brain lesion.'

Other medical writers of authority show that aphasia is characterised not only by purely intellectual deficiency, but by emotional changes and impairment of powers of judgment and discretion. For instance Nielson in *Agnosia, Apraxia and Aphasia* refers to the view of certain writers that in every case of aphasia there is 'a disturbance of the entire psyche'.

MacDonald Critchley in *On the Testamentary Competence of a Patient with Aphasia* says:

'The very nature of aphasic disorder renders difficult any evaluation of the state of intelligence. Certainly in many cases of speech defect, particularly of the sensory or mixed motor and sensory variety, there exists a definite affection of the general intellectual status as well as obvious modifications in what may be termed the total behaviour... In such not only is there an impaired comprehension of what the patient hears or what he sees before him, in writing, but judgment, recollection, calculation and abstract thought are also severely deranged.'

Weisenberg and McBride in *Aphasia* state that in practically every case aphasia

'involves some deterioration in so-called non-language performances, and it also involves changes in the patient's reactions to practical, everyday problems and to matters of social relationship'.

Dr. Geerling expressed the view that owing to its influence on the emotions of the patient (the 'affective disturbance') aphasia might result in impairment of judgment which would affect the will-making capacity. He was also of the opinion that such an incident as the refusal of sexual intercourse might excite more resentment than would be felt by a normal person. Dr. Suzman stated that a cortical aphasia (such as existed in the present case) would affect not only the powers of judgment and discrimination of the patient, but also his character and emotional stability. Dr. Watt agreed that aphasia ordinarily resulted in a change in the emotions and character of the patient and in his general psychological condition: there was a disturbance of some sort of the entire psyche; the patient became emotional and unstable; his reaction to a grievance might be more extreme; it might result in an extreme and unreasonable reaction; there would be impairment to a greater or lesser degree of the patient's normal powers of judgment and discretion.

The condition resulting from aphasia is of course not one of insanity or imbecility in the popular or general sense. Though his intellectual powers are diminished the patient remains sane and, according to Dr. Watt, even in the early stages after the stroke he may retain to a considerable extent his memory and his volition, he may have a sound concept of his own affairs in his mind, and he may be able to grasp fairly complex ideas, such for example as will-making, or divorce, or income from investments, even though unable to convey such ideas to others. This is in conformity with the evidence of Dr. Geerling and Dr. Suzman that an aphasic does not necessarily lose all his powers of rational understanding. It appears to me, further, though the medical witnesses did not touch specifically upon this aspect of the matter, than an aphasic may, and probably in many cases does, realise only too well the impairment of his former powers of speech, of writing, of reading and of following spoken language, and that he may make consistent attempts to recover these lost or diminished powers, for example by undergoing speech training or by having books and papers read to him, or by copying writing.

By way of caution, I should point out here that though the common results or concomitants of aphasia are according to the evidence such as I have described, they are not invariably, or to the same degree, found in all aphasic sufferers. The degree of aphasia varies, and the extent of mental deterioration accompanying it may vary in extent and character according to individual cases. It cannot be said of an aphasic that because he has aphasia he cannot do this thing or that.

According to the experts the degree of impairment of the intellect can only be determined in any individual case by an exhaustive and lengthy neurological examination, such as was never carried out in the case of the deceased. It was accordingly contended on behalf of the defendant that the plaintiff had not discharged the *onus* of proving that the deceased had no testamentary capacity when the disputed will was made. The Courts are, however, almost daily called upon to decide disputed issues of fact without the aid of scientific proof. When that is the case they must take such evidence as is put before them and decide the issue upon the probabilities.

In considering the question whether the deceased had recovered his intellectual faculties to such an extent as to have had testamentary capacity in April 1948, it is necessary first to refer to the evidence, of which a considerable body was led, as to his condition prior to his arrival at Nigel.

The witnesses whose evidence throw most light upon his condition during this period are Dr. Kriek, who examined him while he was at Mossel Bay at the request of the Pensions Department in connection with his application for a military pension; J. A. Shapiro, who saw him on a number of occasions at East London; E. M. Zion and Mrs. Harris, who speak of his state while in the Jewish Aged Home; and Nesbitt, who had him under his care at Avalon. There were other witnesses, namely Major McIntyre and Dr. Hartley, who saw the deceased on a few occasions during the period, but their evidence throws no independent light upon his condition, and I do not think it necessary to say more than that it reinforces the evidence of the four witnesses whom I have mentioned.

The evidence of these witnesses leaves no doubt whatever that the deceased's faculty of speech during this period was extremely limited. He was unable to speak in grammatical or connected sentences. He had a few words at his command, and those which he could utter were often misused. He had great difficulty in remembering proper names and place-names. What he wished to convey had usually to be elicited from him by a series of questions as to what he meant, the correct answer being reached by a process of elimination. A person who knew him well would have less difficulty than a stranger in gathering his meaning from the words he uttered. The evidence of the witness Nesbitt shows that while at Avalon he had a somewhat more extensive vocabulary and greater facility in conveying his ideas than previously and that there was a slight degree of improvement during the course of his stay here; but, throughout his stay Nesbitt had difficulty in understanding him, and his facility of speech fluctuated, so that at times he was according to Nesbitt unintelligible.

He was unable to write spontaneously. Letters would be written out for him in block capitals, which he would copy, but apparently without any comprehension of the meaning of what he was copying.

He was unable to read. Letters received by him would be read to him, the contents being paraphrased for him into simple language by the reader. He liked to have the newspaper read to him, and this led to a slight conflict of evidence as to his ability to comprehend without assistance what appeared in the newspaper. Dr. J. A. Louw, a medical practitioner who attended him for influenza and saw him on three or four occasions at Oudtshoorn, stated that the deceased would point to a headline in the newspaper and indicate that he would want the headlined portion read to him, Dr. Louw drawing the inference that he was 'partially' able to read. In this evidence he is supported by Mrs. Rebecca Lewin, the widow of the deceased's brother, in whose house he was staying. The plaintiff, however, told the Court that the deceased would merely ask for the newspaper to be read to him without indicating any particular article, and that she had no recollection of his ever having indicated that he knew what a particular paragraph was about and wanted it read for that reason.

In view of the positive evidence of Zion, Mrs. Harris, and Nesbitt that during their periods of observation of him the deceased was quite unable to read, it appears to me that Dr. Louw was probably mistaken either in his observation or his recollection or in the inference which he drew that the deceased was able to read the headlines. If he was able to read the headlines he must have been able to read the other matter in the newspaper, yet no witness throughout the case testified that he had ever done so or that he had ever read a book or magazine.

Mrs. Rebecca Lewin testified that on one occasion, during the deceased's stay at Oudtshoorn, he was able, without assistance, to spell out with alphabetical blocks her name, namely, 'Becky Lewin', his own name, and the name 'Mossel Bay'. This, if true, would show a certain degree of ability to comprehend the meaning of the written word. This evidence, however, does not carry conviction to my mind. This witness was obviously resentful of the slur involved, or supposed to be involved, in any suggestion that the deceased was of unsound mind and was inclined to exaggerate his rationality. I was not much impressed with her reliability as a witness. Having regard to the evidence as a whole, I have come to the conclusion that during this period the deceased was totally unable to understand written language.

The evidence of Shapiro, Zion, Mrs. Harris and Nesbitt shows that throughout the period with which I am dealing there was a great impairment of the deceased's ability to understand spoken language. Zion speaks of the necessity of putting everything that was said to him in the simplest language and of hammering away at him until he understood; and this witness states that even after this process he could never be certain that the deceased had understood. Mrs. Harris is to the same effect. Even after his arrival at Avalon Nesbitt found, when trying to teach him to shave himself or to walk, that he was unable to comprehend what was said to him without the aid of ocular demonstration. Throughout the period of his stay at Avalon what was said to him had to be put in very simple language, and spoken slowly so that it could sink in; and it would have to be repeated over and over again.

It is interesting in this connection to refer to the case of Dr. Lordat, which was discussed during the evidence of Dr. Watt. Lordat was a French physician who was interested in the study of speech disorders. In 1838 he suffered a brain lesion which resulted in a severe sensory and motor aphasia; he was unable at

the time to understand what was said to him or to express himself. In 1843, however, he had sufficiently recovered to write a book in which he described his symptoms and the difficulties he had experienced at the time. The speech of others came too fast for him to comprehend it; he 'knew' words but could not use them properly; he used the wrong words, and in trying to correct himself again spoke unintended words; he could not read what he himself had written.

Nesbitt, like Zion and Mrs. Harris, could not be certain that the deceased always understood what had been said to him, even though his replies indicated that he appeared to do so. A comparison of the evidence of these three witnesses shows that though there was some improvement over the whole of the period covered by their evidence, it was not very marked.

The existence of the defects which I have just discussed does not prove that the deceased was incapable of entertaining perfectly rational ideas, and ideas even of some complexity. I have referred to the evidence of Dr. Watt on the point that an aphasic, even in the early stage of his aphasia, may retain to a considerable extent his memory, his volition, and a conception of fairly complex ideas and of his own affairs. I think it is important to ascertain to what extent the deceased retained this power in the early stages after his physical recovery from the stroke and the operations which shortly followed it. I regard this as of importance because his ability to entertain somewhat complex ideas in the later stage of his history was advanced as proof that he was to all intents and purposes then a rational man. A comparison between the early and the later periods will therefore be valuable as throwing some light upon the validity of this contention. When I refer to the early period I mean that prior to the entry of the deceased into the Jewish Aged Home, when he lived at Oudtshoorn, Mossel Bay and East London. Though evidence was not specifically directed to this point, it does show that even at that early stage the deceased retained a considerable memory of his past life, an interest in general affairs, and some grasp of his own business affairs. For instance, while at Oudtshoorn, according to Mrs. Rebecca Lewin, he was able to recognise Oudtshoorn residents whom he had known previously; he would discuss Zionism with his brother; he asked for information about his wife's management of his affairs and disapproved of what she had done in that connection. At Mossel Bay he refused to move into a house which his wife had arranged to rent, because the steps of the verandah were high, it would be difficult to get him in and out of the house, and he apprehended that his wife would be less likely on that account to take him to the beach. While at East London he was able to entertain the idea of a divorce; he was resentful of the sale by his wife of the Westminster farm and of her action in investing, through her father a sum of £1,000, which she had realised by the sale of certain movable property. He was able to take part in a discussion with his brother and his wife of a suggestion that he should be put under curatorship and that the brother and the wife should be appointed curators. His wife discussed with him the question of the rents being received from Leander House, and his insurance policies. Apparently at his request, his wife made out a list of insurance policies, which she posted to a niece for delivery to him after his departure for Cape Town, he at that time apparently having entertained an idea of surrendering some of these in order to reduce his monthly expenditure, for he discussed this suggestion with Zion while in the Jewish Aged Home.

These illustrations gathered from the evidence show that the deceased at that early stage had retained to a considerable extent his memory and his powers of comprehension. No doubt if this aspect had been more directly investigated many more examples would have been brought to light. The facts elicited do not prove, however, that there was no mental deterioration and no psychological disturbance resulting from the cerebral lesion which he had suffered.

There is ample evidence that in addition to the difficulties of speech and of comprehension to which I have already referred, there were other signs of such deterioration and disturbance, some of which are to be found throughout the period up to his departure from Avalon. His memory for recent events was very defective. There was an inability to keep his attention fixed upon the matter being discussed with him for any length of time; after a few minutes his attention would wander, and he might interpose or ejaculate a quite irrelevant remark. This appears from the evidence of Nesbitt as well as that of Shapiro and Zion, though there appears to have been some improvement in this respect during the Avalon period.

Further, although it is clear that he had rational ideas upon a number of matters and was able to take an interest in his own affairs, there is evidence of a lack of insight into those affairs. Both Shapiro and Zion say that he spoke as if he were very wealthy; Nesbitt says that he did not appear to grasp money matters and did not understand what an overdraft was, and that he gave the impression that he always had plenty of money, and was 'mad' to buy a farm. It was as a result of his extravagance while at the Jewish Aged Home that he was eventually placed under curatorship. The desire to go farming while at Avalon appears to me to reveal a lack of insight either into his physical condition or into his financial position, or into the requirements of farming. His persistent smoking is described by Drs. Geerling and Suzman as due to lack of insight into his condition. It was suggested that this may have been due to a deliberate choice to shorten his life rather than give up his enjoyments; but in view of evidence that the deceased was genuinely anxious to improve his conditions and hopeful of doing so this theory does not appeal to me.

There is considerable evidence that during the period which I am now considering the deceased was emotional, easily moved to tears and easily irritated or annoyed; and that he was capricious and changeable in his likes and dislikes. There is no doubt on the evidence of his son, as well as that of his wife, that he was hypersexual in the early stages; and it appears from Nesbitt's evidence as to an occasion when Mrs. Nesbitt went to his room, and he said to Mrs. Nesbitt 'I want woman, I want woman', that this condition continued into the Avalon period and was accompanied by lack of restraint which is characteristic of disturb-

ances resulting from aphasia of the type from which the deceased was suffering.

It was contended on behalf of the defendant that the evidence as to the deceased's mental condition while he was at the Jewish Aged Home and at Avalon revealed a man who had sufficient intellectual capacity and such a grasp of his own affairs as to be fully capable of making his will. Reliance was placed, more particularly, upon the contents of certain letters and other documents which were put in as evidence. The evidence shows that when the suggestion of a curatorship was made in 1947, the deceased being then in the Jewish Aged Home, he made it a condition of his consent to the application that an independent accountant, who was to be an ex-serviceman, would be appointed as curator and that the curator would pay him £30 per month for his maintenance, out of income, and if insufficient, out of capital.

In regard to this argument it is interesting to notice that according to Zion, it was only after he had spent about three weeks hammering away at the idea of a curatorship that the deceased appeared to understand, and agreed. When the papers had been drawn for the necessary application to Court the attorney dealing with the matter (Mr. Stern), the prospective curator (Mr. W. Goldberg) and another gentleman called upon the deceased, apparently in order to obtain his formal agreement, and Mrs. Harris was present during part of the interview which followed. Mrs. Harris states that she was present for about an hour, that Stern and Goldberg were trying to explain to the deceased something about property, but while she was present they were able to obtain no response from him. This evidence suggests a doubt whether the conditions stipulated by the deceased emanated quite spontaneously from him. It may well be that they were suggested to him as being in his own interest or for his protection, and that he agreed or appeared to agree. Mr. Stern, however, though in Court instructing counsel for the plaintiff, was not called, and I am prepared to accept that the deceased did understand the nature of the application which was being made and the conditions which were ultimately embraced in the order.

Then there was certain correspondence between the deceased and his curator while the deceased was at Avalon. The letters of the deceased would, according to the evidence, be blocked out for him by someone else, and he would either copy them in block letters with his own hand, or type them out on a typewriting machine. Some of the ideas contained in these letters emanated according to Nesbitt spontaneously from the deceased without having been suggested by the person who drafted the letter; for instance the first three paragraphs of the letter Exhibit 15, the financial enquiries in Exhibit 16, and probably the whole of the draft letter Exhibit 18. Nesbitt states that the ideas and wishes embodied in these letters would be laboriously extracted from the deceased in the manner to which I have already referred.

It was urged that the ideas involved in the application for curatorship and in the deceased's stipulations in that connection, and also in the contents of the letters to the curator, were of so complex a nature that if the deceased could comprehend them he must have been sufficiently rational to have possessed testamentary capacity. In my view, however, the concepts involved are not particularly complex, and I do not regard it by any means as impossible that they should be grasped and understood by a man suffering from the kind of mental impairment which according to the expert medical witnesses results from a cerebral thrombosis.

Considerable importance was also attached, more particularly by Dr. Watt, the expert witness called by the defendant, to the fact that while at Avalon the deceased had come to a decision that his son Gerald, then a student of soil conservation at the Witwatersrand University, should proceed to South America on a course. This incident is referred to in the evidence of Nesbitt. It appears that a students' tour to the Argentine, to last a month or two, had been arranged, that the son was anxious to go on the trip but had insufficient funds (see Exhibits 17 and 37), and that Nesbitt approached the deceased for his consent to the trip and also for his financial assistance. The consent was given, and funds were provided out of moneys which the deceased did not require for his subsistence at Avalon. It was contended that this showed a power of exercising judgment and discretion such as indicated a normal and rational man. It does not appear to me, however, to involve any great degree of judgment or discrimination. The deceased was quite capable of grasping the idea of an educational trip overseas for his son, and the decision only involved assent to the two suggestions that he should go, and that he should be given money for the purpose out of funds which the deceased himself did not need. The agreement to the suggestion may indeed only be an instance of the suggestibility which according to the medical evidence is a characteristic of this form of mental disorder.

Generally, the evidence in so far as it bears upon the nature and complexity of the conceptions which the deceased was capable of entertaining while at the Jewish Aged Home and at Avalon does not seem to me to reveal any great advance upon those which he held in the earlier stage, as revealed by the examples which I have set out above.

The evidence as to the condition of the deceased during the whole of this period between his emergence from the Groote Schuur Hospital and his departure from Avalon gives me the general impression that there was a gradual improvement both in his physical and mental state. He had weaned himself—at least to some extent—from dependence upon drugs; he had learned to walk with the assistance of rails erected by Nesbitt, to shave himself, and to use a typewriter with one finger; he had regained some power of movement in his paralysed arm; and his general health was better. Broadly the picture drawn by Nesbitt is of a person somewhat less helpless, both physically and mentally, than the person described by Shapiro, Zion and Mrs. Harris. This may be partly accounted for by the fact that at Avalon he was in sympathetic hands, and happy in his surroundings. The improvement over the whole period of two-and-a-half years was, however, in my view by

no means spectacular. I am fortified in this view by the opinion of Dr. Geerling that over the whole period covered by the evidence of the witnesses whom he heard, there was no marked improvement in mental capacity, and by that of Dr. Suzman, who also heard the evidence led for the plaintiff, that there was no significant improvement in his powers of understanding.

Let me now turn to the period of the deceased's stay with the defendant after his departure from Avalon and arrival at Nigel. If I am to believe some of the witnesses called for the defendant, the improvement in the condition of the deceased within the space of a week or two can be described as startling. According to the witness Truss, the manager of the hotel at which the deceased stayed with the defendant and his wife until his removal to hospital for the operation for appendicitis, in the course of a discussion on religion on the day of his removal to hospital, the deceased was able to say 'If no Roman Catholic, no Jewish religion, no English religion, no Afrikaans religion, if we had one way to bow down to God, and one common approach, there would not be so much money making'. According to Mrs. Stella Lewin, wife of the defendant, in her evidence in chief, at this discussion he said that he did not think there should be any churches, the main thing was how you felt in your heart towards your fellow man and God, and continued 'you can worship God by looking at the hills as well as you can in any church'. If these statements are to be accepted the deceased must have acquired in the fortnight since he had left Avalon a truly remarkable degree of coherence and fluency. When he was seen, however, by Dr. van Staden on 12 or 13 March, by Dr. Segal on and after 14 March and by Lockett the attorney who drew the will, about a month later, his method of speech as described by these witnesses was not materially different from that described by Nesbitt. It is difficult indeed to imagine the remarks deposed to by Truss and Mrs. Stella Lewin as having come from the person interviewed by Drs. van Staden and Segal, and by Lockett.

Other witnesses called by the defendant gave evidence as to the manner in which the deceased spoke during this period. Of these the evidence of the witnesses E. C. Sawyer and Sister Tong seem to call for particular notice. Sawyer saw him early in April, a few days after he had left the Nigel Hospital, and discussed with him the advisability of surrendering some of his insurance policies in order to reduce his expenditure. At this interview, according to Sawyer, though the deceased spoke very slowly and was occasionally at a loss for a word, his sentences were perfectly connected and there was no difficulty in understanding what he said. similarly Sister Tong, who attended him in the Florence Nightingale Nursing Home between 29 April and 2 May, said that he spoke in normal language, very slowly and deliberately, and he stumbled over words, but he used complete sentences including all the connecting words. These two witnesses were quite disinterested and gave their evidence well. Their contact with the deceased was, however, comparatively slight, and they were asked to recollect conversations which had occurred more than a year before they entered the witness box. Their evidence as to the fluency and coherence of the deceased's conversations is at variance with that of other witnesses called for the defence, in particular Drs. Segal, Myers and van Staden and Lockett. It appears to me that their recollection of the manner in which the deceased conversed was probably faulty. As to the matter of the deceased's conversations with them, he gave Sister Tong certain information of a simple nature about himself, and his ability to do so is not in my view inconsistent with the existence at that time of mental deterioration of the character associated with aphasia. The main subject matter of his conversation with Sawyer, namely the advisability of surrendering certain of his policies in order to reduce his expenditure and so conserve his income, was undoubtedly of a more complex character; but he had been considering this matter, according to Zion, even while in the Jewish Aged Home, so that the ideas involved would be familiar to him, and as I have pointed out, according to the expert witnesses an aphasic may entertain conceptions of some complexity. The substance of what the deceased said could have been conveyed in comparatively few words to a man, such as Sawyer, who was conversant with the problems which present themselves to holders of insurance policies.

In addition to evidence of the manner in which the deceased spoke, a great body of evidence was led for the defence to show that the deceased was able to understand everything that was said to him, that apart from the fact that he was a cripple and had a speech defect he had normal tastes and inclinations and lived a normal life, and that he had a complete grasp of his own business affairs; in short, that he was to all intents and purposes a completely rational man.

I do not propose to discuss all of this evidence in detail; it is I think sufficient to say of it, generally, that it is to a large extent beside the point, in view of the evidence of the expert witnesses to which I have already referred that in cases of motor and sensory aphasia the patient may retain to a considerable extent his memory, his volition, his understanding of his own affairs, and his power of rational understanding. Proof that an aphasic has behaved rationally in a number of directions therefore does not establish that he is completely normal and has recovered from his aphasia. It is also in evidence that the condition is variable, and that the patient's power of comprehension is greater at some times than at others. It appears to me, further, that a number of the witnesses called for the defence have, consciously or unconsciously, exaggerated the rationality of the deceased during this period. In giving evidence as to information conveyed to them by the deceased or statements made by him they have probably not distinguished between words actually uttered by him and their own interpretations of his remarks or the interpretations thereof made by the defendant or his wife, or information volunteered by the latter. Mrs. Stella Lewin stated that she and the defendant would often volunteer information to strangers in order to save the deceased embarrassment caused by his difficulty of speech.

(To be continued)

VERENIGINGSNUUS : ASSOCIATION NEWS

BARAGWANATH MEDICAL SOCIETY

JULY MEETING

Mr. E. S. Brawn presented a middle-aged Bantu male who complained of swelling in the right side of the neck and hoarseness of the voice for three months, and inability to open the right eye. Examination revealed bilateral hard glands in the tonsillar region, much larger on the right. In addition there were glands beneath the sternomastoid which were fixed. There was complete ophthalmoplegia of the right eye; the patient was able to distinguish light. The optic disc was normal. Laryngoscopy showed a swelling on the right side, thought to be an indentation of the pyriform fossa from without, deforming the vocal cords and pushing them over to the left. Radiological examination showed normal sinuses but there was erosion of the right posterior clinoid process. Bronchoscopy was negative and an angiogram showed no abnormality of the cavernous sinus and carotid artery. All other investigations were negative.

Mr. Brawn discussed the ocular palsies and demonstrated involvement of the maxillary infra-orbital branch. There was no involvement of the fronto-nasal or lachrymal nerves. No proptosis was present. He therefore felt that it was unlikely that the nerves were involved far back in the cavernous sinus but rather by upward growth of glands in the pterygomaxillary fossa extending upwards to the inferior orbital fissure. Such a spread would explain involvement of the infra-orbital branch of the maxillary nerve in the floor of the orbit and the third, fourth and sixth nerves at the apex of the orbit. Biopsy of a gland revealed a squamous cell carcinoma and he thought the case to be one of epilaryngeal carcinoma with secondaries in the glands.

Dr. L. H. Horwitz presented a case of a young adult Bantu female complaining of pain in the chest for seven days and cough productive of a whitish non-offensive sputum. One month prior to admission she had a premature labour, the infant dying shortly after birth. There was no previous history of cough and no deaths in the family from tuberculosis. Physical examination revealed a very distressed patient breathing rapidly, with a tachycardia of 132, numerous moist rales over both bases and mid-zones and a pleural rub in the right axilla. The white cell count was 9,000 per c.mm. Repeated examinations of sputa, which later became greenish, were negative for tuberculosis but on one occasion a Friedlander's organism was isolated.

Penicillin therapy was given for one week with no response. Streptomycin 0.5 gramme every six hours given subsequently produced some favourable response although the physical signs remain unchanged. The radiological examinations showed diffuse mottling over both sides of the chest, which persisted despite therapy.

The blood-pressure varied between systolic 100-80 mm. and the diastolic 80-60 mm. Hg. The tachycardia persisted and the patient died three weeks after admission.

Dr. F. P. Reid discussed the differential diagnosis. He suggested that the radiological appearances and the course of the disease were compatible with tuberculosis but the apparent lack of response to streptomycin and the negative sputa were against it. Other causes of radiological appearances of discrete mottling were discussed. Fungal and yeast infections, the reticuloendothelioses, fibrosis of unknown etiology, eosinophilic infiltrations, virus pneumonitis, chorionic epithelioma, multiple emboli from the legs, and septic emboli from the uterus were mentioned.

Dr. J. Higginson discussed the post-mortem findings which showed a resolving bronchopneumonia. A large number of organisms was isolated but a predominant type could not be determined. Dr. Horwitz concluded by stating that bronchopneumonia of this type was not uncommon in this hospital. The etiology was difficult to determine. A falling blood pressure of systolic 90-80 mm. Hg. and diastolic 60 mm. Hg. often preceded the other signs of peripheral circulatory failure by several hours and often one day. The treatment for this complication was Methedrine, serum and intravenous glucose or glucose-saline depending on the urinary chlorides. One had often felt hesitant of this form of therapy in a collapsed patient with moist lungs. Immediately after death lung puncture to determine the predominant organism with subsequent typing against the various antibiotics might be a helpful procedure in the future treatment of similar cases.

A further case of unresolved pneumonia was shown; two separate bronchoscopies revealed multiple diphtheroid plaques. The patient was subsequently readmitted with multiple abscesses in the right upper lobe and Dr. Horwitz suggested that the basic lesion was an atelectasis due to these diphtheroid plaques, the breakdown of lung tissue being the result of secondary infection.

THE BENEVOLENT FUND

The following contributions to the Benevolent Fund during July 1950 are gratefully acknowledged:

Votive Cards: *In Memory of*:

Mr. F. J. Geggie *by* Dr. George O'Neill Waddington.
Miss Douglas *by* Joy and Ian Macgregor.
Capt. W. D. Hare *by* Dr. Vernon Brink, Dr. A. H. Tonkin.
Mrs. M. Stanton *by* Dr. J. W. Harris.
Dr. A. W. Louw *by* Dr. K. Cunningham (amount received March 1950).

Total Amount Received from Votive Cards:	4	4	0
Services Rendered to:			
Dr. H. Gottschalk *by* Dr. F. Petersen.			
Total Amount Received from Services Rendered:	10	10	0
Donations:			
Dr. E. F. Moon		5	0
The Medical Officers of Health (State Medicine) Group of the Medical Association of South Africa	10	10	0
Natal Coastal Branch Members	114	6	6
Dr. H. Renton	1	1	0
Legacy Estate Late Dr. C. M. Murray	200	0	0
	£340	16	6

PASSING EVENTS

BARAGWANATH MEDICAL SOCIETY

A clinical and clinico-pathological meeting will he held on Monday 25 September 1950 at 8 pm. in the Nurses' Training School, Baragwanath Hospital, Johannesburg.

* * * *

Dr. B. Krikler, formerly of Muizenberg, has returned from overseas and is now in practice at 707 Cavendish Chambers, Jeppe Street, Johannesburg.

BRITISH JOURNAL OF TUBERCULOSIS AND DISEASES OF THE CHEST

Dr. Philip Ellman is succeeding Dr. Clifford Hoyle in the editorship of the British *Journal of Tuberculosis and Diseases of the Chest*, after the publication of the July issue, and he will be assisted by an Editorial Board which, in addition to Dr. Clifford Hoyle, comprises Mr. T. Holmes Sellors (London), Dr. A. Brian Taylor (Birmingham) and Professor Cameron (Edinburgh).

Dr. Clifford Hoyle succeeded the late Dr. L. S. T. Burrell in 1938 and has, therefore, edited the journal for 12 years.

REVIEWS OF BOOKS

OXIDATION-REDUCTION POTENTIALS

Oxidation-Reduction Potentials in Bacteriology and Biochemistry. By L. F. Hewitt, Ph.D., B.Sc., F.R.I.C. (Pp. 215 + viii. With illustrations. 20s. 6th edition. 1950.) Edinburgh: E. & S. Livingstone Limited.

Contents: 1. General Considerations. 2. Practical Methods. 3. Measurement of pH. 4. Systems of Biological Interest. 5. Metabolic Cycles and Chain Reactions. 6. Bacteriological Applications. 7. Chemotherapy and Antibiotics. 8. The Polarograph 9. General Conclusion.

Oxidation-reduction potentials have come to play an important part in biology. This book gives to the student an easily understandable account of the meaning and measurement of the potential of reversible oxidation-reduction systems, and to the more experienced worker the technical and mathematical details on which the measurements are based.

These measurements are essential to a proper understanding of the orderly and co-ordinated succession of reactions which occur in the respiration of bacterial and animal cells. These systems and their co-ordination in aerobic and anaerobic respiration are fully discussed. Oxidation-reduction reactions are the essence of the function of many well-known constituents of the cell; many pigments, enzymes and co-enzymes constitute two stage oxidation-reduction systems and are essential for biological oxidations. The actions of some of them are not reversible, and therefore do not lend themselves to this method of measurement, but it appears that some such non-reversible systems may in the natural surroundings inside the cell work in such close association with reversible systems that they become amenable to this type of investigation. The close relation of the vitamins of the B group to respiratory enzymes is well established. An account is given of the extensions of this work to include the reactions of vitamins C and E, of some hormones and of antibiotics.

These extensions of the subject are fully treated in the new edition, which is printed in a very attractive style. There is a full bibliography. It will be of particular value to the bacteriologist, but all biologists will find much of interest and value in it.

OBSTETRICS AND GYNAECOLOGY

Combined Textbook of Obstetrics and Gynaecology for Students and Practitioners. Edited by Dugald Baird, B.Sc., M.D., D.P.H., F.R.C.O.G. (Pp. 1,411 with 594 illustrations, 30 in colour. 70s. 5th ed.) Edinburgh: E. & S. Livingstone Ltd. 1950.

Contents: 1. Anatomy of the Genital Organs. 2. Physiology of the Reproductive System. 3. Management of the Normal Reproductive Function. 4. Pathology of Pregnancy. 5. Gynaecology. 6. Gynaecological Operations. Appendix. Index.
Contributors: Dugald Baird, B.Sc., M.D., D.P.H., F.R.C.O.G.; A. S. Duncan, *D.S.C.*, M.B., Ch.B. (Ed.), F.R.C.S. (Ed.), M.R.C.O.G.; E. Chalmers Fahmy, M.B., Ch.B., F.R.C.S. (Ed.), F.R.C.P. (Ed.), F.R.C.O.G.; Stanley Galbraith Graham, M.D., F.R.C.P. (Ed.), F.R.F.P.S.; R. W. Johnstone, C.B.E., M.A., M.D., M.R.C.P., F.R.C.S. (Ed.), F.R.C.O.G., F.R.S.E.; Robert J. Kellar, M.B.E., M.B., Ch.B., F.R.C.S. (Ed.), F.R.C.P. (Ed.), F.R.C.O.G.; J. M. Munro Kerr, M.D., LL.D., F.R.F.P.S., Hon. F.R.C.O.G.; I. Leitch, M.A., D.Sc.; T. N. McGregor, M.D., F.R.C.S. (Ed.), F.R.C.O.G., F.R.S.E.; H. R. MacLennan, M.D., F.R.F.P.S., F.R.C.O.G.; Robert McWhirter, F.R.S.E., M.B., Ch.B., F.R.C.S. (Ed.), D.M.R.E., F.F.R.; W. M. Millar, M.D., D.Psych.; W. I. C. Morris, M.B., Ch.B. (Ed.), F.R.C.S. (Ed.), F.R.C.O.G.; G. Leslie Purser, M.A. (Cantab.), F.R.S.E.; James Young, *D.S.O.*, M.D., F.R.C.S., F.R.C.O.G.

The new edition of this well-known textbook, the contributors of which are all eminent obstetricians and gynaecologists from the Scottish Universities, makes a welcome appearance.

The book conforms to its previous pattern in that both obstetrics and gynaecology are combined in one volume, the two subjects being linked by a connecting chapter.

Certain inevitable changes have taken place in this edition. New chapters dealing with stillbirth, infant mortality and psychological factors in obstetrics and gynaecology have been added. Special attention has been given to the chapter on physiology of pregnancy.

A pleasing feature of this work is the emphasis on the preventative and physiological approach to the subjects. Referring to foetal mortality, Dugald Baird states in his preface: 'It has become apparent that many foetal deaths are associated with forms of "physiological failure" rather than definite pathological processes. Thus the goal is no longer prevention of death, but the attainment of efficient physiological reproduction.'

The chapter on psychosomatic medicine in relation to obstetrics and gynaecology is an interesting one. This aspect of medicine has become prominent in recent years; and while it certainly has a place in modern obstetrics and gynaecology, care should be taken that too much emphasis is not placed on the psychological aspect. Referring to shock therapy, W. N. Millar makes the following comment: 'Unless, however, there is definite evidence of a psychotic reaction present, such drastic procedures should be regarded as most dubious and empirical, and resorted to as a last measure.' This point is worthy of emphasis.

Under the capable editorship of Dugald Baird, the contributors to this edition have produced a book which is modern in every sense. The illustrations, many of which are in colour, are lucid and helpful. Most of the chapters conclude with a short bibliography.

Whilst specifically written for students and medical practitioners, this is a textbook which should also prove of value to the post-graduate student specializing in obstetrics and gynaecology. It can be thoroughly recommended.

THE SULPHONAMIDES

The Sulphonamides. By F. Hawking, M.D., and J. Stewart Lawrence, M.D., M.R.C.P. (Pp. 390 + viii, with 46 illustrations, including 12 plates (seven in colour) and 17 tables. 42s.) London: H. K. Lewis & Co., Ltd. 1950.

Contents: 1. Introduction. 2. Chemistry of the Sulphonamides. 3. Estimation of the Sulphonamides in Body Fluids. 4. Action upon Bacteria. 5. Acquired Resistance of Bacteria to Sulphonamides. 6. Pharmacology. 7. Mode of Administration. 8. Choice of Compound. 9. Streptococcal and Staphylococall Infections. 10. Pneumococcal Infections. 11. Meningococcal Infections and Meningitis. 12. Gonorrhoea and other Venereal Diseases. 13. Infections of Urinary Tract. 14. Intestinal Infections. 15. The Treatment of Wounds and Burns. 16. Gas Gangrene. 17. Peritonitis. 18. Dermatology. 19. Diseases of the Ear, Nose and Throat. 20. Diseases of the Eye. 21. Miscellaneous Infections. 22. The Sulphonamides in Prophylaxis. 23. Toxic Reactions caused by Sulphonamides. 24. Renal Obstruction due to Sulphonamides. 25. Sensitation to Sulphonamides. 26. Toxic Effects upon the Blood and Marrow. 27. Other Toxic Effects of Sulphonamides. Bibliography. Index.

The sulphonamides have been in clinical use for about 15 years. While only a few are prescribed to-day, an enormous number of compounds has been investigated, and hundreds of publications have appeared in journals. Manufacturers have bombarded doctors with literature on their particular brands, much confusion resulting from the multiplicity of trade names.

No outstanding developments in this type of chemotherapeutic agent have occurred in the last few years, so that the subject may now usefully be reviewed. This task has been excellently performed by the authors of the book under review. They provide a thorough account of the chemical nature, mode of action, pharmacological actions and clinical uses of sulphonamides, of the lesser known compounds, but especially of those in common use. The literature from all sources, including that which was not available during the war years, has been carefully consulted, and personal experience by the authors included.

It is pointed out that further advances will depend on the discovery of compounds with lower toxicity, or more advantageous regarding absorption, metabolism, or excretion. Since 1941, when sulphadiazine was introduced, compounds more active than this sulphonamide have not been added. Practitioners should prescribe sulphonamides by official names, although they may still favour a particular brand, and doses should be prescribed in grammes, not in terms of tablets. A full list of trade, chemical and official names is given. Doctors have been known to prescribe the same sulphonamide by another trade name, when attempting to avoid sensitization by a particular sulphonamide.

The question of resistance to sulphonamides is fully considered, including available knowledge regarding the nature of the mechanism underlying this phenomenon. Prolonged but ineffective concentration of the drug may lead to drug-

resistance with no further clinical response. This has been common in the treatment of infective endocarditis, in which condition antibiotic drugs are now favoured.

Attention is drawn to the exaggerated importance that has been attached to the relatively low concentrations of certain sulphonamides in the cerebrospinal fluid, which are generally identical with the levels of uncombined or active drug in the plasma and therefore equally effective.

Each of the sulphonamides now used has certain suitable properties, and much information is available in this book on the choice of compound. Treatment is described fully, with charts, tables and illustrations. Toxic features are considered in detail. It is interesting that sulphanilamide may cause acidosis by inhibiting carbonic anhydrase.

There is an extensive bibliography, itself a valuable feature of the book. Although this may not be the last word on sulphonamides, there is here available a comprehensive review of the whole subject, well produced and well documented.

ELECTROCARDIOGRAPHY

Electrocardiography: Fundamentals and Clinical Application. By Louis Wolff, M.D. (Pp. 187, with 110 figures. 38s. 3d.) Philadelphia and London: W. B. Saunders Company. 1950.

Contents: Part I: The Basic Principles of Electrocardiography. 1. Electrical Phenomena associated with Muscle Contraction, Electrical Properties of the Cell Membrane, Depolarization, Dipoles. 2. Volume Conductors, Unipolar and Bipolar Leads. 3. Deflections which represent Depolarization, Intrinsic and Intrinsicoid Deflections. 4. Vector Representation of Dipoles. Vector Summation. Anatomy and Physiology of the Heart. 5. The Electrical Effects of the Simultaneous Excitation of Two Muscle Masses in a Volume Conductor. 6. The Electrical Field Around the Heart. 7. Repolarization, Primary and Secondary T Wave Changes. 8. The Precordial Electrocardiogram. 9. Unipolar Limb Leads Standard Limb Leads. 10. The Einthoven Equilateral Triangle Hypothesis. Instantaneous Electrical Axis, Ventricular Gradient. 11. Right Bundle Branch Block. 12. Left Bundle Branch Block. 13. Left Ventricular Hypertrophy. 14. Right Ventricular Hypertrophy. 15. The Effect of Muscle Injury on the Electrocardiogram. 16. Current of Injury. Blocking of the Depolarization Wave. 17. Myocardial Infarction. *Part II: Introduction.* 18. The Normal Electrocardiogram. 20. Bundle Branch Block. 21. Left Ventricular Hypertrophy. 22. Right Ventricular Hypertrophy. 23. Coronary Heart Disease. Myocardial Infarction. 24. Myocardial Infarction and Bundle Branch Block. Persistent S-T Segment Displacement, Arborization Block. 25. Pericarditis. Pulmonary Embolism. 26. Short P-R Interval with Abnormal QRS Complexes (Wolff-Parkinson-White Syndrome). Index.

Throughout this book the author has made a successful endeavour to simplify, as far as possible, the complicated problems of the subject.

At the beginning of the book that bugbear of the student, the electro-physical phenomena of cardiography, have been dealt with in masterly fashion, amply illustrated by simple diagrams. With the essential preliminaries (the electrical behaviour of the muscle strip) behind him, the reader is conducted by easy stages until he is able to approach the details of the praecordial electrocardiogram with confidence.

Here the clinician will note with pleasure that, while the practical aspects of the electrical position of the heart are treated adequately, the manifold combinations of rotation of the heart in three dimensions, the minutiae of which become such an academic maze in certain publications, are glossed over to a considerable extent.

The second half of the book is devoted to clinical electrocardiography, and the practising physician will admire the essentially clinical approach and the stress laid on the pitfalls inherent in making a diagnosis to the exclusion of the history of the case.

The cardiographic patterns associated with left and right ventricular hypertrophy are neatly set out and the resemblance of such tracings to those obtained in myocardial infarction is demonstrated.

These important aspects and many more, too numerous to detail, are ably dealt with in this work.

At a time when physicians are endeavouring to familiarize themselves with the new unipolar lead method, there is no doubt that this beautifully bound book, with its many excellent diagrams and tracings, is an important contribution to the subject. It can be recommended with confidence to any student of electrocardiography.

THE DIABETIC LIFE

The Diabetic Life. Its Control by Diet and Insulin. By R. D. Lawrence, M.A., M.D., F.R.C.P. (London). (Pp. 238 + xi. With 18 illustrations. 14th Edition. 10s. 6d.) London: Messrs. J. & A. Churchill Limited.

Contents: 1. A Comparison of Normal and Diabetic Metabolism. 2. The Causation and Pathology of Diabetes and the Discovery of Insulin. 3. The Symptoms of Diabets. 4. Diagnosis and Blood Sugar Tests. 5. Principles of Treatment by Diet and Insulin; The Line-Ration Scheme; The Qualities and Action of Different Insulin Preparations. 6. Actual Treatment by Diet and Insulin; Who Needs Insulin?; Diet Treatment of Mild Cases by Weighed Diet; Treatment by a Simple Unweighed Diet; Treatment with Protamine Zinc Insulin. 7. Treatment by Soluble Insulin Alone. 8. Further Problems in Diabetic Management; Future Progress and Prognosis; Is Insulin a Cure?; Different Ideals and Methods of Treatment; The Omission of Insulin; Insulin Sensitivity and Resistance. 9. Insulin Injections. 10. Hypoglycaemia and its Treatment; Endogenous Hypoglycaemia. 11. Treatment of Coma and Precomatose Conditions. 12. Treatment of Children. 13. Control of Variations and Complications in Treatment; Pneumonia and Other Acute Infections; Thyroid Disorder and Diabetes; Pregnancy and Diabetes. 14. Operations and Gangrene: Perforating Ulcers and Infection of Bone; Carbuncles. 15. Miscellaneous: Oedema and Dropsy; Diuresis; Pigmentation of the Skin; Pancreatic Disease; Vitamins; Contra-indications. 16. The Choice and Management of Diet. 17. Diabetic Recipes. 18. The Essentials of a Diabetic Education; Care of the Feet. 19. The Five-Gram Scheme and Food Tables: Lists of Dangerous Foods, 'Extras,' etc.; Average Weight Tables; Equivalent Tables; Detailed Examples of Line Diets.

The fourteenth edition of Dr. Lawrence's book has been brought thoroughly up to date both as to the recent concepts of the disordered metabolism of diabetes and in the use of the newer insulin preparations in its treatment.

Dr. Lawrence intended this book for use both by the practitioner and by the patient himself, and the book undoubtedly fulfils this dual purpose. During the past 25 years it has been used throughout the world and has been translated into four foreign languages. In it the general practitioner will find everything that he need know in the day-to-day handling of diabetic patients and in the treatment of the emergencies that may arise. In discussing diabetic coma it is emphasized, however, that this emergency is best handled in hospital, where facilities for biochemical tests and skilled treatment are available. Dr. Lawrence does not believe in the very heavy insulin dosage which has been recommended for diabetic coma, and it is difficult to dispute the validity of the arguments he advances. Certainly for anyone other than the specialist in diabetes, and particularly where coma is not profound, the rather conservative method advocated by Dr. Lawrence undoubtedly is the treatment of choice.

The basis of the dietetic management of diabetes remains as before, the 'Line-Ration Diet Scheme' which was introduced with the first edition of his volume and which has proved one of the most satisfactory schemes yet evolved for the guidance of the diabetic. It is in this section on dietetics that the patient will find the book of the greatest value, and there are few diabetics who would not benefit from reading those sections expressly intended for the patients.

In spite of the volume of valuable information the book contains, its size has remained small, and the clear, concise style makes it very easy to read and understand. It can be most highly recommended both to the medical practitioner and to his diabetic patients.

MEDICINE FOR NURSES

Lectures on Medicine to Nurses. By A. E. Clark-Kennedy, M.D., F.R.C.P. (Pp. 288 + viii, with 28 figures. 15s. 6d.) Edinburgh: E. & S. Livingstone, Ltd. 1950.

Contents: 1. The patient and his Disease. 2. Acute Rheumatism. 3. Old Age. 4. Nephritis. 5. Diseases of the Lungs. 6. Tuberculosis and Syphilis. 7. Acute Abdominal Pain. 8. Chronic Abdominal Pain. 9. Anaemia and Jaundice. 10. Fever, Throats, Glands and Rashes. 11. Disorders of the Mind. 12. Paralysis. 13. Unconsciousness. 14. Cough, Constipation, Sleeplessness and Pain. 15. Diet and Diabetes. 16. Vaccines and Serum Therapy. 17. Chemotherapy and Radiotherapy. 18. The Desperately Ill Patient. Index.

Hand in hand with the medical man's ever-increasing knowledge of the mysteries of life, the demands on the nursing profession daily become greater and more exacting. It is not surprising, therefore, that each season brings a new crop

of books, of varying quality, on the many different aspects of nursing.

In spite of the undoubted excellence and importance of many of these new publications, Dr. Clark-Kennedy's *Lectures on Medicine to Nurses* stands alone. It is not his masterly grasp of his subject, nor his almost uncanny knowledge of a student nurse's psychology, which makes this book unusual. One expects nothing less from the Dean of the Medical School of the London Hospital; but it is the excellence of the writer's prose, the imaginative delicacy and intelligence of his thought-sequences and the charm of his personality which, seeping through the printed page, compel one's sympathy and almost breathless concentration from the very start, and make this book remarkable.

It was a happy thought on Dr. Clark-Kennedy's part to publish his series of 18 lectures, originally delivered to second- and third-year student nurses at the London Hospital, and so make them accessible to everyone; for these lectures will not only profit the nurses who study them; they will prove of inestimable value to all those who are responsible for the training of nurses. Though the author's way of teaching may be considered unorthodox by some, basically, it constitues the ideal relationship between teacher and pupil. 'I intend, as far as possible, to teach you principles which you can understand and remember, and deliberately to avoid filling up your minds with facts that you will memorize and forget. I do know that it is difficult to think; it is always easier to learn things by heart. But take my advice, try to think out the answer for yourself. As a result of that mental effort, it (the knowledge) will be permanently registered on your mind.'

This is a book that should be in an easily accessible place, on every Sister-Tutor's bookshelf, and with every sympathy for the student nurse, hag-ridden by too little time and the bogey of examinations, it is nevertheless recommended, not as a textbook, but as a guiding light and an inspiration.

GYNAECOLOGY

Gynaecology. By H. H. Schlink, M.B., Ch.M. (Sydney), F.R.A.C.S., F.R.G.S.; C. L. Chapman, *D.S.O.*, V.D., Med. des Epid., M.B., Ch.M. (Sydney), F.R.C.S. (England), F.R.C.S. (Edinburgh), F.R.A.C.S.; G. G. G. L. Stening, E.D., M.B., B.S. (Sydney), F.R.C.S. (Edinburgh), F.R.A.C.S., M.R.C.O.G. (England); F. N. Chenhall, M.B., B.S. (Sydney), F.R.C.S. (England), M.R.C.P. (Ireland), F.R.A.C.S. (Pp. 650. With 198 figures and 19 coloured plates. Second revised edition. 1949. 67s. 6d.) London and Sydney: Angus & Robertson, Limited.

Contents: 1. Anatomy. 2. Physiology. 3. Disorders of Function. 4. Symptoms found in Gynaecological Disease. 5. Specific Infections of the Female Urogenital Tract. 6. Localized Inflammation. 7. Extra-Uterine Pregnancy and Abortion. 8. Injuries, Prolapse and Displacements of the Female Generative Organs. 9. Tumours and New Growths of the Female Genital Organs. 10. Malignant Growths of the Female Generative Organs. 11. Examination of the Patient and Ethical Problems. 12. Operative Gynaecology. 13. Post-Operative Complications: Their Prevention and Management.

There is a multitude of good books, well produced and well illustrated, to guide the undergraduate student through the gynaecological maze. This volume is a useful addition to the literature and will probably be particularly welcome to Australian students.

Dr. Schlink points out in his preface that 'the whole of the book has been reviewed, any accepted new principles in gynaecology since 1939 have been incorporated, and especial attention has been paid to the newer forms of treatment.

The chapters on anatomy, endocrinology, disorders of menstruation, etc., have been re-arranged and that on gynaecological urology has been enlarged. The chapter on endometriosis has been largely rewritten. The chapters on cancer, operative procedure and post-operative complications have been thoroughly revised, and a new chapter on virilism added.'

It is to be regretted, however, that in the section dealing with pregnancy tests the volume incorrectly refers to the Xenopus test reported by Shapiro and Zwarenstein in 1933 as the Hogben test. No doubt this error of acknowledgment will be remedied in future editions which the volume undoubtedly deserves to achieve.

MEDICAL GYNAECOLOGY

Medical Gynecology. By J. C. Janney, M.D., F.A.C.S. (Pp. 454 with 108 figures. 55s. 3d. or $6.50. New, Second Edition.) Philadelphia: W. B. Saunders Company. 1950.

Contents: History and Physical Examination. 2. The Patients' Complaints. 3. Physical Findings. 4. Tests and Special Examinations. 5. Gynecologic Treatments. 6. Socio-medical Problems in Gynecology. 7. Irradiation or Operation. Bibliography. Index

Prof. J. C. Janney has been able to cull from the experience of 25 years in preparing this attractive volume which now reaches its second edition.

The book should be an invaluable guide for the student as well as the young practitioner in dealing with the human female, of whatever age.

The volume is distinguished by useful tables summarizing differential points of importance and a multitude of well-selected illustrations adds much to the clarity and the effectiveness of the author's presentation.

Its practical orientation is revealed by attention to such points as are dealt with in chapter 57, viz. emotional, economic and social factors, and the very thorough account of contraception in chapter 58 will be invaluable to the young practitioner who will also find much to guide him in this book to the solution of the psychological problems with which the newly-married will approach him.

The section on artificial insemination draws attention to the moral and legal aspects raised by this difficult problem. The medical practitioner, however, will find much of practical guidance in the precautions he should take to protect himself and a useful specimen of a consent form is quoted from the literature.

This attractively printed volume is a welcome addition to the shelf of the undergraduate student as well as the medical practitioner.

CORRESPONDENCE

NOT SO FUTILE RADIOLOGICAL PROCEDURES

To the Editor: Dr. Berman in his letter in the *Journal* of 29 July 1950, admits performing routine chest screening before barium meal examination. It is more than probable, therefore, in spite of his unwillingness to 'presume' normality of a chest which 'screening' showed to be clear, that Dr. Berman does not take a routine film of that chest lest the clinician be lulled into a false sense of security by such a report!

In other words Dr. Berman's 'practice' in this regard conforms to accepted radiological diagnostic technique, for the simple reason that despite this limiting qualification it has not been found futile, in the same way and for the same reasons that sinus screening has also not been found futile.

I quite agree with G.P. in his letter published on 12 August 1950, that radiology is today a science of experts and as such can safely be left with them.

H. Hirschson.

Union House,
Queen Victoria Street,
Cape Town.
15 August 1950.

To the Editor: It is extremely difficult to know how to discuss Dr. V. Berman's opinions of the value of fluoroscopy of the sinuses. In his letter, published in this *Journal* on 17 June, he categorically states 'it is recognized by authoritative radiologists that fluoroscopic examination of the sinuses is a futile procedure', and 'I know of no X-ray department overseas which takes upon itself the responsibility of commenting upon sinus pathology from a screen examination; nor can I find any reference to this method in the literature'. Yet, when he is referred to the statement indicating the value of preliminary screening of the nasal sinuses, in a standard textbook of radiology used by all students for the D.M.R., he writes (his letter in the *Journal*, 29 July) 'I have always been aware of this statement by Dr. Twining'.

It is quite clear from Dr. Berman's letters that he does not appreciate what information, of positive value, can be obtained by the procedure he, alone, considers to be futile.

For his benefit it appears necessary once more, to inform him, emphatically, that an opinion is formed indicating only that the fluoroscopically accessible sinuses are clear, or not, on screening, and not whether they are normal. The function of the radiologist is not to usurp that of the clinician.

Dr. Berman's asseveration 'it is recognized by authoritative radiologists, etc.', should not be accepted as reflecting the true picture of the radiological position in this regard.

What can only be accepted is documentary proof of this affirmation. His unsupported word is hardly enough, for at this moment his contention is a truth by assertion only.

In my own experience, covering the examination of thousands of chests, the routine fluoroscopy of the sinuses has resulted in the detection of innumerable cases of unsuspected infection. The unveiling of such infection has benefited both the patient and the clinician. I have taught the value of the procedure so severely deprecated, and so harshly criticised, by Dr. Berman. It has been interesting to read that Dr. C. J. B. Muller, one of my pupils for the D.M.R. and, until recently, one of my assistants, has shown figures (in this *Journal*, 22 July) to support his own experience with it.

Surely figures are truth by fact, resulting from observation and experience. They must refute Dr. Berman's assurances that 'fluoroscopic examination of the sinuses is a futile procedure'.

Verifying an hypothesis by the test of facts is a very different process from that of supporting an hypothesis because one fancies it. An assertion can only become valid and substantial when evidence to support it is forthcoming.

Dr. Berman's condemnation appears to be based on the belief, or idea, that the inability to detect finer changes, by screening, does not warrant the radiologist to look for grosser ones. His admission 'at best one could say that this or that antrum appears to be opaque' (17 June) is a contradiction of his condemnatory attitude. Dr. Berman's criticism, in spite of its efforts, cannot get away from experience, nor can it dissolve the whole extent of the proven experience of others, by the aid of *a priori* ideas.

That sinus screening, as an adjunct to fluoroscopy of the chest, is not futile, is proven to the hilt by past radiological practice and experience. The facts of experience point unhesitatingly, and with justification, to the routine screening of the accessible nasal sinuses, when a patient is referred for radiography of the chest.

In reply to 'G.P.' whose letter appeared in this *Journal*, 12 August, I believe that the implacable and relentless criteria of 'the science of experts' (his words) will not permit astonishment, and emotional pleading, to masquerade as judgment. Such criteria provide the only basis acceptable for scientific criticism. It is only by intelligent discernment that fact can be separated from fiction.

J. N. Jacobson.

Barclays Bank Buildings,
St. George's Street,
Cape Town.
21 August 1950.

KIDNEYS: DO THEY DROP?

To the Editor: Dr. Campbell Begg in the *Journal* of 19 August, discredits renal ptosis, but also takes radiologists to task for reporting this condition. 'Ptosis' is derived from the Greek πτωσις meaning 'fall' or 'dropping', and radiologically the term is usually used to indicate dropping of the kidneys, in an erect subject, from their situation in the supine subject. A kidney which occupies a low position with the patient recumbent is not ptosed, for, if ptosed or dropped, from where has it dropped?

The inclusion or omission of reference to ptosis in urography as determined by the above procedure, will depend largely on the views of the doctor referring the case, and as many doctors ask for it, Dr. Campbell Begg's plea to omit ptosis from X-ray reports will depend on their reception of his argument. It is of little concern to the radiologist if the doctor reads too much into the term ptosis, and there is nothing to stop the radiologist from stating that 'in the erect position the degree of renal ptosis is within normal limits', if that is so in his opinion.

Dr. Campbell Begg mentions that 113 out of 396 cases were reported to have renal ptosis. This represents 29%. If the term ptosis is used in the above sense, the number of cases must be considerably higher. Indeed, some degree of renal ptosis in the erect position is the rule rather than the exception.

E. E. Faerber, M.B., D.M.R.

16, Leicester Mansions,
207, Jeppe Street,
Johannesburg.
24 August 1950.

SO-CALLED 'STEVENS-JOHNSON SYNDROME'

To the Editor: I was interested to read the report in this *Journal* on a case of recurrent non-specific urethritis, associated with respiratory catarrh, stomatitis, conjunctivitis, and an erythematous vesicular eruption of the legs and forearms[1] diagnosed as Stevens-Johnson syndrome.

I doubt, however, whether the term 'Stevens-Johnson Syndrome'[2] as usually described, is strictly applicable. If, indeed, an eponym must be employed, perhaps Behcet's disease[3-7] with its 'triple symptom complex' of genital, oral and ocular lesions would be the more appropriate diagnosis in this case. The case is strikingly similar in many respects—especially in regard to chronicity and recurrence over a period of years—to those cases of Behcet's disease described by Katzenellenbogen.[6, 7]

The Commission on Acute Respiratory Diseases[8] and others[5, 9] emphasize, with regard to Stevens-Johnson syndrome, that the eponym is without historical justification.

Robinson and McCrumb[5] in a critical analysis and comprehensive review of Behcet's disease; Reiter's disease with its urethritis, conjunctivitis and arthritis; 'Stevens-Johnson Disease'; and ectodermosis erosiva pluriorificialis, conclude that there are so many points of similarity between them that they are probably all variants of the same disease entity, erythema multiforme, excepting possibly the so-called Reiter's disease. They suggest the term 'muco-cutaneous ocular' syndromes to cover the whole group.

These authors also emphasize that, according to the present-day concept, all the above entities differ decidedly from the original descriptions, and that until the specific aetiology is discovered, there is no justification for the classification of these symptom groups as distinct entities.

Klauder[10] classifies erythema multiforme into symptomatic (due to infection, drugs, sera, etc.) and idiopathic types, the latter believed to be due to a virus.

It is possible that the symptomatic form may also be of virus origin, the causal factors 'tilting the delicately balanced virus-host relationship' in favour of the virus.

REFERENCES

1. Helman, J. (1950): *S. Af. Med. J.*, **24,** 688.
2. Nellen, M. (1947): Lancet, **2,** 326.
3. Behcet, H. (1937): Dermat. Wchnschr., **105,** 1152.
4. Editorial (1950): Brit. Med. J., **2,** 1479.
5. Robinson, H. M. and McCrumb, F. R. (1950): Arch. Dermatol. Syph., Chicago, **61,** 539.
6. Katzenellenbogen, I. (1950): Arch. Dermatol. Syph., Chicago, **61,** 481.
7. Katzenellenbogen, I. (1946): Brit. J. Dermat., **58,** 161.
8. *Commission on Acute Respiratory Diseases* (1946): Arch. Intern. Med., **73,** 687.
9. Sir Henry Cohen (1947): *Personal communication.*
10. Klauder, J. V. (1937): Arch. Dermatol. Syph., Chicago, **36,** 1067.

Maurice Nellen.

316, Geneva House,
Parliament Street,
Cape Town.
25 August 1950.